HUMAN RACES

Frontispiece. Urinary chromatogram of a 33-year-old Chinese woman. The arrow points to the area occupied by BAIB (β amino isobutyric acid) an amino acid frequently excreted by individuals of Asiatic origin. For details see Figure 8 and Chapter III.
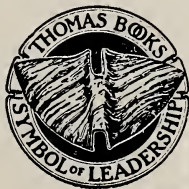
HUMAN
RACES

By

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PREFACE

Ten years ago Carleton Coon, Joseph Birdsell and the present author collaborated on a little book entitled *Races: A Study of the Problems of Race Formation in Man.* In it we eschewed the then traditional anthropometric measurements and morphological ratings, and compiled no formidable catalog of human racial groupings. We were interested in one central problem—how human races came to be.

When we wrote *Races,* the mere mention of race was still uncomfortable to many, that soon after the tragic excesses of the Third Reich. But we were not concerned with notions of racial superiority or inferiority. We were writing about races in man, how they arose and how they changed, as they are changing still.

Races was venturesome for its time, a time when the concept of a "pure race" was still tenable, and when scholars still wrote of fixed, static and unchanging races, incapable of genetic change. But the tempo of discovery soon passed us by. Critically investigated, using the new tools of biochemical genetics, human races proved capable of more rapid change than the most optimistic guess would have warranted. Directions of natural selection within race populations, a subject we had speculated about, proved most varied, and at the same time susceptible to exact measurement. With renewed interest in human raciation, problems of human differentiation have been newly tackled. The entire field of Geographical Medicine, a newcomer among the disciplines, has added vital meaning to the study of race.

Human Races now is a very different book from what *Races* (1950) was. It is one man's product, both Coon and Birdsell being busy with their own investigations and their own publications on race. At the same time, *Human Races* is the contribution of many investigators, the results of a most active decade of race-

research. Some of these investigations have been reprinted in *Readings on Race* (1960), a companion source-volume to the present work, and therefore not recapitulated in detail here.

Human Races is an attempt to describe what race is, and the mechanisms of racial differentiation in man. It will, I hope, help to dispel the antiquated notions of three "original" races, of the persistence of racial types, and of the role of undirected chance in bringing about racial differences. In their stead, I trust will emerge the contemporary picture of man's genetic response to local selective factors, the constantly changing nature of the natural populations we call races.

While a more complete listing of indebtedness is given later in this book, I would like to thank Lois Conklin (who has lived through three books with me), Laura Newell, who has drawn illustrations, located references and corrected errors, and Dr. Lester W. Sontag and the Fels Fund for both tangible and intangible support.

<div align="right">STANLEY M. GARN</div>

ACKNOWLEDGMENTS

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I

THE NATURE OF RACE

Nearly three hundred years ago, Carolus von Linnaeus, the great naturalist and taxonomist, set up his famous classification of living things. When he came to man, Linnaeus properly assigned man to the order *Primates* on the basis of numerous and fundamental biological similarities. To the genus that contained man, he gave the traditional Latin name *Homo*. And, having weighed the evidence for and against several species of man, von Linnaeus assigned all living forms of mankind to one species within the genus *Homo*, as *Homo sapiens*.

Today, we know far more about man than Linnaeus did. We have recovered from the Pleistocene deposits of Java and China fossil species of *Homo* that are quite distinct from *Homo sapiens*. We have come to study many groups of living men quite unknown in Linnaeus' time. Beyond the simple descriptions available to the Swedish taxonomist, we have precise anthropometric measurements, data on blood groups, the haptoglobins and many measures of biochemical functioning. Although there are some traits in which different human groupings show little overlapping, living mankind clearly constitutes a single polytypic species. Fossil non-sapiens hominids no longer exist, and we are all *Homo sapiens*, as assigned by Linnaeus.

But, within this single species which now covers the habitable globe there are many discrete groupings, some so clear-cut as to be obvious to the least-trained observer, and others less easily distinguishable except after intensive study. These groupings, differing greatly in size and taxonomic status have commonly been lumped under the single term "race." Thus, some so-called races are grossly distinct by all of the tests we now have and use, while other groups called races differ in smaller degree, in the averages

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of certain measurements, in the proportions of discrete traits, and in the frequencies of such biochemical differences as the several blood groups.

In addition to races defined by zoologists, anthropologists and human geneticists, human groupings of various kinds have been dignified or designated by the term "race." Race has been equated with language, and that is the sole useful meaning of the "Aryan" race. Race has been identified with religion, as in the case of the Jewish "race," which in reality comprises a number of discrete populations, some quite unrelated to the others. National groupings have frequently been called races, especially in periods of intensive nationalism. While at times linguistic groupings and biological races may coincide and while religions or even national boundaries may delimit race-populations of various sizes, language, religion and national affinity are hardly measures of race. Race is a biological concept and races are biological units. Races, moreover, are natural units and not artificial assemblages created by selecting "types" out of a population.

A century ago many scholars believed that all human groups could be explained in terms of a few "original" races, usually numbering three. Many attempts were made to explain known human populations as mixtures, in varying degree, of these hypothetical original races. In the days when all dark-skinned peoples were accepted as "Negroes," when all straight-haired people were accepted as members of the "yellow" race, an original set of but three races satisfied the data and fitted the assumptions. Yet, it is now obvious that such a working hypothesis is untenable. No possible combination of Negro, Mongoloid and White could produce the Australian aboriginal. No such combination could explain the American Indian. The blood group distributions in Melanesia, Micronesia and Polynesia could hardly be explained in terms of Negro-Mongoloid-White admixture. In fact, there is little reason to believe in a system of but three original races, that but three original races ever existed.

In Europe too, scholars formerly postulated the one-time existence of a limited number of "pure" races, which through admixture, gave rise to the complex situation we see from Finland to North Africa today. As proof of their hypothesis, they pointed

to the occurrence of *individuals* exemplifying the characteristics attributed to the hypothetical pure races. From blue-eyed, long-headed, light-haired men and women they inferred the "Nordic" race, and from various combinations of features, found in individuals, other ancestral "pure" races were similarly deduced.

Yet, while the individuals themselves, as "types" clearly exist, the inference that they recapitulate ancestral strains can be challenged. As Edward E. Hunt, Jr. (1959) has demonstrated, the individual "types" are merely chance combinations of the genetically independent traits. Since the traits are independent, and "segregate" out separately, such types as may be found in a population prove nothing about the appearance of ancestral groups. Blue eyes and blond hair are no more proof for an original Nordic race than red hair and freckles point to an original Rufous race, or short stature and heavy beards to a race of Trolls.

In its heyday, however, the approach to race as a series of types, led to racial typing of *indivduals,* with rather remarkable results. Various members of a single family were often assigned to different "races," and three brothers or three sisters could be typed as belonging to as many different "races" (Fig. 1).
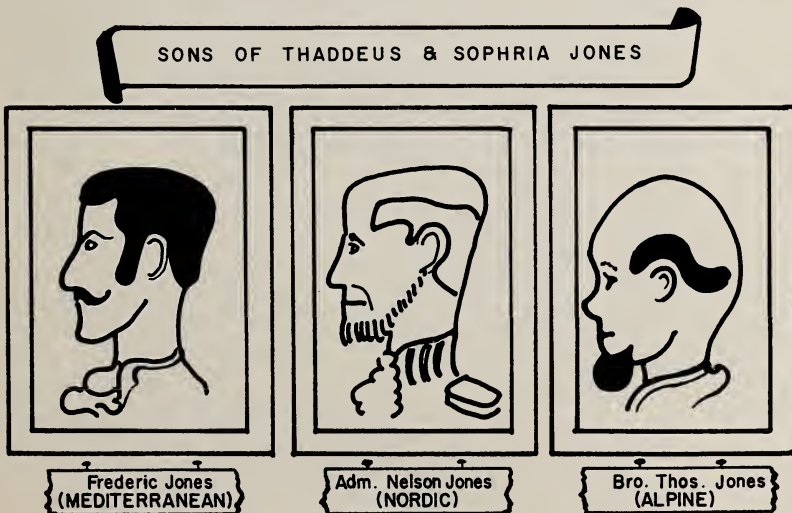
Fig. 1. The concept of race as type. In this approach, where individuals are classified as to appearances, brothers may be assigned to different "races."

THE CONTEMPORARY APPROACH TO RACE

The contemporary approach to race in man, as in other living forms, is eminently simple. It is a return to the basic principles of classification, to the days before hypothetical original races were postulated, and populations were analyzed in terms of varying proportions of different racial "types." Yet the contemporary approach to race stems from population genetics, where a race is viewed as a breeding population, neither more nor less. A race in man, as in any living form, is a *population,* a population of men, women and children, of fathers, mothers and grandparents (Fig. 2). Members of such a breeding population share a common history, and a common locale. They have been exposed to common dangers, and they are the product of a common environment. For these reasons, and especially with advancing time, members of a race have a common genetic heritage.

Fig. 2. The concept of race as population. In this approach, emphasis is on the biological race or population *isolate.*

Such a definition, race as identical with the breeding popula-
tion, transcends history. One race, or one breeding population
may go back 20,000 years, or even more. The Central Austra-
lians, and possibly the Andamanese, can claim such antiquity,
with little or no admixture. Another race, as for example the
American-Colored, may have been formed by admixture in recent
memory, but it has equal claim to racial status. A third race, far
smaller than either of the two mentioned, but no more recent than
the American Colored, is the genetically and geographically iso-
lated population of Pitcairn Island. If there is hesitation at calling
the Pitcairners a race, how large must a population be to be a
race? If admixture, both known and recent, provides a stumbling
block to acceptance, how about the probable incorporation of
Neanderthals into the European population, and the certain addi-
tion of late-Paleolithic survivors as late as 8,000 years ago?

From the standpoint of taxonomy, that is classification, how a
race was formed is irrelevant. A race is a race is a race whether it
goes back unchanged for six millennia, or whether it resulted from
admixture after 1850. Our preoccupation with recent history may
make us view the Neo-Hawaiian race with hesitation, but how
about races whose mixed origins barely antedate the written
record? In similar fashion, there is no number test for race. The
Bushmen of South Africa, totalling perhaps 25,000, are quite as
real a breeding population as the American Colored, who number
some 15 million.

Moreover, there is increasing evidence that races do change in
their genetic makeup, even in the absence of admixture. The
sickle-cell gene, for example, has been on the increase in Africa
ever since slash-and-burn agriculture was introduced. With drain-
age, mosquito control, DDT and antimalarial drugs, the sickle-cell
gene will decrease in generations to come. Differences between
races once thought to go back to remotest antiquity, now appear
to be genetic responses to environmental conditions. Recognizing
that changes do occur within races, that races are not fixed in
their genetic makeup, our attention has shifted from the simple
existence of races, and the description of the differences, to the
mechanisms that bring these changes about.

OTHER NAMES FOR RACE

So far in this book the term *race* has been used to designate natural human populations, the sum of which constitutes the species as a whole. To be technical, the "race" is the taxonomic (that is classificatory) unit immediately below the species. Thus it is that the single species *Homo sapiens* comprises a number of races, of varying magnitude, as will be discussed in the next chapter. The majority of physical anthropologists, taxonomists and geneticists agree with this usage, holding to the term race, despite misuses that have occurred in the past.

A minority of workers, however, prefer other terms for the same taxonomic units. The word race, they argue, has been ill-used by politicians and demagogues, by Madison Grant, Lothrop Stoddard, de Gobineau, and by Adolph Hitler. Some "races," as Sherwood L. Washburn has pointed out, never existed. Like the "Nordic" race of Germanic fame, they were hypothetical formulations that had no bearing in fact. It would be advisable, according to one argument, to use a new term for race, free of unfortunate historical associations, and which would mean exactly what we want it to mean.

Various euphemisms for race have been suggested, among them "variety," and "division." The geneticist Hans Kalmus (1950) prefers the word "strain," a term used by animal breeders and plant geneticists. Ashley Montagu (1951) in much of his recent writings, has championed the term "ethnic group" as a substitute for the familiar word "race."

However, these substitute terms have defects of their own. A botanical "variety," such as the dwarf Forsythia, is not equivalent to a polymorphic human population. A strain or breed is an artificial line, for the most part maintained by selective breeding. The term "ethnic group," useful in its original sense of a cultural group, creates confusion when equated with biological races, the more so when a list of "ethnic groups" proves virtually identical with other lists of races!

To be sure, it is not tremendously important what term we use for race, and here we may recall Shakespeare's comment on the rose. A race-population remains a race-population whether called breed, variety, strain, or ecar, which is simply race spelled back-

wards. A newly-minted, specially-coined word might be free of unfortunate associations yet would contribute little to the amelioration of racial prejudice. Under the circumstances we might as well continue to talk about races, devoting our attention to spelling out the exact taxonomic category under consideration.

THE STUDY OF RACE

The real question, of course, is why we study race, why we are interested in raciation in man, and why we expend valuable time and considerable money in the investigation of such seemingly esoteric subjects as the blood groups of the Idaho Basques.

For some the existence of races is a challenge, as with mountains that beg to be climbed simply because they are there. In much the same vein, an isolated race, unsullied and unstudied, still beckons with romantic appeal. As with Richard Burton, there are always some who will brave dangers and risk their own fortunes to be the first to visit and describe a previously unstudied population.

Still others are interested in taxonomy, that is classification. And to have a complete taxonomy, that is a complete classification, all human populations must be seen and studied. We are far from a complete taxonomy for man: part of Africa, much of Asia and a surprising portion of South America is still to be investigated with care. In the meanwhile, new race-populations are springing up, and these too merit analysis.

Another reason for studying race is phylogenetic. How are races related to each other, and which races arose from others? In some cases we have historical data to guide us as with the Cape Colored, and the American Colored, the Ladino and the neo-Hawaiian. Even so, serological data are necessary in order to complete our knowledge as to proportions entering into admixture. In other cases we own mere educated guesses. Clearly the American Indians stem from Asiatic Mongoloids but when and where? Are the Bushmen ancient, going back to the earliest origins of Africa, or are they merely desert-adapted Negroes? Conversely, are the Zulu and Bantu themselves evolved Bushmen?

These latter problems bring us to the more intriguing reasons for investigating race, including the question of how races and

particular races came to be. Why are Negroes dark, and are there similar explanations for the peoples of Southern India, and the Melanesian Islands? Why do Asiatics have straight, coarse black hair and sparse beards? Why are the Papago fatter than the Navaho in the same region? Why are the Basques so peculiar in their Rh blood group distribution? Why are the Pygmies pygmy, the Blackfoot Indians tall, and why are the Aleut short?

Why are certain genetic diseases limited to some groups and absent in others? Since such diseases are deleterious and subject to natural selection, what explains the continuance of sickle-cell disease in Africa, Favism and Mediterranean anemia in Southern Europe, or Kuru in New Guinea? And turning from disease to physiology, are the Eskimo cold-adapted and are the natives of Dakar or Brazzaville adapted to both heat and high humidity as would be reasonable to suppose?

Such questions as these are no longer excursions into the unanswerable. Within the past decade we have seen some of them explained, and others close to understanding. How races came to be is no longer a philosopher's conundrum, and the peculiarities of particular races come closer and closer to comprehension. The distinctive characteristics of every race may now be understood in terms of the special environments in which they have lived.

The study of race bears a personal attractiveness to us. This is our species, and as men we are inevitably fascinated by man. Now, quite suddenly we are in a position, as many investigative fields come to maturity, to answer the fundamental questions that will lead to a more complete understanding of the different races of mankind.

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II

GEOGRAPHICAL, LOCAL AND MICRO-RACES
RACE, "RACE" AND *RACE*

THE original edition of *Races* published in 1950, contained a listing of thirty living race-populations in man. In the very same year, in his *Genetics and the Races of Man,* William C. Boyd described a total of six races: however, one of them was both hypothetical and extinct. Though Boyd (1958) has more recently augmented his list of races, raising the number to thirteen, we are obviously dealing with different orders of magnitude.

Other listings of human races have run the numerical gamut. A century ago some workers divided mankind into but two races, a straight-haired race and a woolly-haired race. Most physical anthropologists have described from twenty to fifty distinct races, though some of these races refer to individual "types" and not to breeding populations. Specialists, working with particular areas of the world have been even more generous in their race-assignments, granting fifty or more for Europe alone (cf. Coon, 1950).

Faced with such a perplexing situation, such a range of numbers of races, it is customary to refer to "lumpers" and "splitters." Lumpers, among taxonomists, are those who group a number of distinct varieties into one broader, larger category, explaining that the differences are too trivial to warrant so complex a taxonomy. Splitters, on the other hand, take the microscopic rather than the macroscopic view holding that any distinct variety merits attention.

But the situation in regard to man is not simply a matter of lumping or splitting: rather it is due to an overly elastic use of the term *race.* For some workers, such as Boyd (1950) *races* are identical to geographically-delimited collections of races. Practically, Boyd's "races" are identical with the "stocks," "divisions,"

"primary races" or "major races" as defined by previous workers. To other human taxonomists, however, particularly those influenced by population genetics, a *race* is a *population*. Inevitably, therefore, equating "race" with population-collections results in a smaller number of races, while restricting the term race to actual populations results in a far larger number of races. It is as if the term regiment were variously employed to refer to platoons, squadrons, brigades and armies.

One expedient would be to coin a new set of words for taxonomic units smaller than the species. One might have the *species*, then the *stock*, then the *breed*, then the *variety*, etc. But there are dangers in seting up a completely new terminology, as mentioned in the last chapter. The most practical suggestion is one made by Bernhold Rensch, the German systematist, in 1929. He uses the term *geographical race*, to describe the broad, geographically-delimited population collections, and the term *local race* to refer to race-populations themselves. As Ernst Mayr (1950) puts it "this system facilitates communication without encumbering nomenclature."

While *geographical races* and *local races* adequately distinguish Bushmen, for example, from Africans in general, or Navahos or Hopi Indians from the broad category of Amerindians, these two terms are not quite enough to fit all of the data. The population of Oslo is genetically distinct from the population of Helsinki, yet neither is a true breeding population, a genetic isolate. Salerno and Padua are distinct in many respects, yet there is no fence about Salerno, and no moat surrounding Padua. Here Dobzhansky's term *microgeographical race* comes to our assistance in delineating statistically-distinct populations which cannot be delimited as circumscribed breeding-populations. However, and with apologies to Professor Dobzhansky, the term *micro-race* will be used throughout this book (instead of micro-geographical race) simply because it is less confusing, less likely to be confounded with *geographical race*.

Geographical races, local races and micro-races, these do not encumber nomenclature. Whosoever uses them is immediately aware of the fact that he is dealing with race, the taxonomic unit immediately below the species. These terms facilitate commu-

nication, in that they explicitly state which taxonomic unit is involved. With respect to geographical races, there is no plethora of them. As race-collections they certainly do not exceed ten. Of local races there is obviously a multiude numbering surely into the hundreds. The Navaho, Hopi, Zuni, Pima, Papago, Cocopa, Haida, Salish . . . these are a few of the local races among the American Indians. And micro-races, in densely populated areas of Europe and Asia—these run into the thousands, each hamlet being genetically somewhat distinct from the others.

Clearly, the one term race is not enough for us to use. By being more explicit we gain clarity and lose confusion.

GEOGRAPHICAL RACES

The *geographical race* is the largest of the three categories of races and encompasses (in each geographical region) the other two. For a geographical race is by definition, a geographically-delimited collection of similar races (Fig. 3). To a large extent the geographical races of mankind coincide with the major con-

Fig. 3. A geographical race—a collection of race populations, separated from other such collections by major geographical barriers.

tinents, and are therefore identical with *continental* races, as the term is used by Boyd and others. However, geographical races may also be spread over major island chains, as is evident in the Pacific today.

The existence of geographical races is due, of course, to the great geographical barriers, chief among them oceans, that formerly limited the expansion and migration of local races and protected them from the introduction of different genes. Thus, in pre-Colonial South America, there was little or no gene-introduction from either Africa or the Pacific. Gene flow in and out of South America was funneled through the narrow isthmus of Central America. The great sub-continent of Australia also represents a situation where geographical race and geography coincide, due to water barriers all around, and no major tradition of navigation and sea travel.

However, the continents marked out in different colors on the map do not perfectly delimit geographical races, whereas the geographical barriers to human migration do. Africa is separated from Europe, and its own northern region, by great ranges of desert, scarcely inhabited by a few wandering tribes, and by the Atlas mountains. South of the Sahara and through Africa to its southernmost tip there is one geographical race, comprising a very large number of local races, whereas North Africa is racially confluent with the Near East and Europe.

Similarly, the eastern limit to the geographical race inhabiting Europe is in Western Asia, in the scarcely inhabited uplands, and not coincident with conventional continental divisions. However, the high and uninviting mountains that mark the Tibeto-Indian border on the maps have long restricted population interchange to a thin trickle. The facts of geography, the mountain ranges, the deserts and the oceans have made geographical races by fencing them in.

Within each geographical race the individual populations resemble each other more or less. In the aggregate, resemblances within geographical races are far greater than those between them. However, intra-population differences are still great, especially taken trait by trait or gene by gene. In each geographical race there are tall populations and short populations, heavy-set

groups and linear groups. Certain human differences transcend geographical race, and are more meaningfully distributed with respect to climate or disease (Chapter VII).

A geographical race is a collection of populations whose similarities are due to long-continued confinement within set geographical limits.

LOCAL RACES

In contrast to geographical races which are geographically delimited population collections, *local races* correspond more nearly to the breeding populations themselves. Whether isolated by distance, by geographical barriers or by social prohibitions, local races are totally or largely endogamous, and the very small amount of gene-flow ordinarily comes from contiguous and related local races (Fig. 4.).

The Bushmen of South Africa are one example of a local race where the territorial limits are defined, and where breeding has

Fig. 4. A local race—a breeding population adapted to local selection pressures and maintained by either natural or social barriers to gene interchange.

been confined almost exclusively to the local race itself. The several native local races of Australia also typify the situation, as do the Ituri-forest Pygmies. Though the latter have contributed wives to the taller Negroes around them, gene-flow appears to be largely one-way, and the Ituri-forest Pygmies constitute a true breeding population to the present day.

Clear-cut local races such as these are largely independent evolutionary units, and as such are of particular interest. Other examples of local races include the Yemenite Jews, isolated reproductively from their Arab neighbors, and from other Jewish populations for millennia. Whereas the Yemenite Jews, now being absorbed into the Europeanized population of Israel, have religious affinities with the European, North African, Kurdistani and Oriental Jews, their status as a separate local race held for thousands of years.

As a further example of local races, one may consider the various Eskimo populations widely spread from Greenland across the Arctic to Alaska, the Aleutians and Siberia. Each has been separated from the other for millennia. It is questionable whether one Greenland Eskimo got to Alaska in the last five-hundred years. As to the Aleut, despite their proximity to Alaska, well under 1% of Alaskan genes have found their recent way into Umnak, Atka, or the Pribilofs.

Local races are most easily identified where populations are relatively small, and there is little doubt as to their limits as indicated by geographical separation or by cultural prohibitions on marriage outside of the group, as with the several Gypsy populations of Europe. Local races can also be delineated, though less neatly in the populous areas of the world. By way of example, the demographic populations of Northwestern Europe and Southern Europe share markedly different histories and are, on the whole, quite distinct. The former population, relatively late to expand, has done so following the discovery of the New World and the subcontinents of the Pacific and has poured into these territories. Thus it is that North America, New Zealand and Australia constitute territorial extensions of Northwestern Europe from a racial point of view, while Central and South America is

more of an extension of the breeding population of Southern Europe.

The Northwest Europeans, though constituting a smaller taxonomic unit than the European Geographical race, are not as neat a population as those mentioned earlier in this section. Numerous special problems interpose themselves, the problem of differential migration (who migrated?) and differential selection in the new and at least temporarily hostile environments. Moreover, even among the stay-at-homes, there are local differences, as shall be mentioned shortly under micro-races.

Nevertheless, it is the local race that we view and measure somewhat more easily when the numbers are small, whereas the geographical race represents more of an abstraction. The population as a unit-of-study is identical with the local race, and becomes increasingly more difficult to investigate as it becomes less easy to delineate.

MICRO-RACES

For much of the world today, as in Europe and Eastern Asia, neat local races are hard to come by. Except for a few populations in Europe such as the Basques, or the Lapps, one cannot define a local race by an ethnographic survey. A man from Berlin marries a woman from Stuttgart, her brother lives in Hamburg and has married a Dane from Copenhagen, whose sister now resides in the Finnish University city of Turku (but is on leave in Cleveland).

Nevertheless, there are very real differences in the genetic makeup of cities, and continual changes in the frequencies of various genes in either the north-south, or the east-west direction. Some of the differences are so apparent that we can divide Europe into a series of local races, Northwest European, East Baltic, Alpine, Mediterranean, etc. Other differences are more subtle, visible to the trained observer, or detectable by the serologist or biochemist from his data.

Regional differences in Europe are in part due to ancient settlement patterns, and to the local perservance of local races. Even though the genetic insularity of the old city-states has long been breached, propinquity is still a very real determinant of mating. "International" marriages, especially in the academic and

professional classes, should not blind us to the fact that marriage, or mating, is a mathematical function of distance. With millions of potential mates, the male ordinarily choses one near at hand. In fact, there is a third kind of genetic isolation. In addition to geographical isolation, as between continents, and cultural isolation, as between local races, there is isolation by numbers. The denser the population, the more nearly the boy marries the girl next door (Fig. 5).

This latter phenomenon, which somewhat deflates the picture of the romantic human male, and allies him more nearly with the field mouse or mosquito (which have similar mating ranges) has the effect of maintaining micro-races. It will be eons, at the present rate, before Copenhagen is no longer different, genetically,
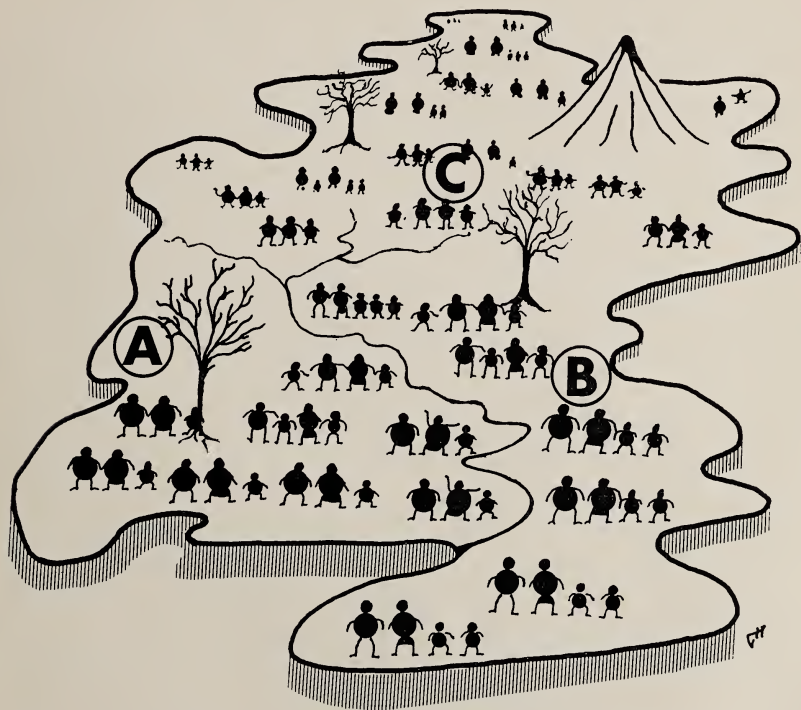
Fig. 5. Micro-races. In a densely populated area local races may not be demonstrable, yet biological distance may maintain regional differences as in the centers A, B and C shown here.

from Oslo or Stockholm, or Venice from Naples or Rome. Further-
more, local selective factors will continue to be at work. Thus,
one genotype will be favored in this city and another in that,
maintaining and even exaggerating the genetical differences that
now exist between micro-races.

TAXONOMY AND RESEARCH ON RACE

Given geographical races, local races and micro-races, there
may appear to be some question as to their relative importance.
From one point of view, geographical races may seem to be of
greatest interest. Geographical races are large, and there are so
few of them. From another point of view local races may be
favored. After all, local races are natural populations (not col-
lections of convenience); they are the basic evolutionary units
and they can be studied in divers ways.

Actually, the importance of these successive taxonomic cate-
gories depends very much on the problem at hand. By way of
example, the ancestry of the American Indians, and their relation-
ship to Asiatics focuses attention on two geographical races.
Differences between Europeans and Asiatics with respect to blood
group B, the Diego factor, or the Rhesus-negative gene, again
involve geographical races. The Polynesians, and possible ex-
planations for their polymorphism, necessitate attention to con-
tiguous geographical races, in Asia, in Melanesia and Papua, and
in Australia.

At the same time, the diversity of populations within each geo-
graphical race introduces problems of its own. If we are to com-
pare Amerindians to Asiatics, with an interest in common origins,
which Amerindians and what Asiatics shall we compare? Shall
we used weighted average values, which may be biased by par-
ticular populations, or try to select prototypical Amerindians and
Asiatics in which case our comparisons obviously reflect the
populations selected for use?

In contrast to geographical races, local races are both easier to
define and simpler to investigate. Within the course of a year
it is possible to measure and blood-type all living Aleut. It is
possible to obtain a fair sample of all known Bushman bands.
Moreover, with local races, we are interested in local selective

factors; diet, disease and environmental stresses. Local races, therefore, commend themselves to the investigation of evolutionary forces. The degree of out-marriage can be determined in assaying the role of "drift." Survivors can be studied to determine possible directions of selection. With local races inhabiting a fixed territory, and where their neighbors can be examined as well, the role of admixture may be quantified. Local races, therefore, offer the maximum opportunity for evolutionary studies.

With micro-races, our human material approaches more nearly the demographic population rather than the natural or race-population. Micro-races are not delimited by geographical or even tribal barriers to gene flow. Nevertheless micro-races offer numerous opportunities to investigate the mechanisms of differentiation. In Saudi-Arabia, as in the more populous parts of Africa, the incidence of malarial infestation may be related to local differences in the frequency of the sickle-cell gene. In European cities it is possible to investigate differential survival in the face of smog, a problem we have begun to consider in Donora, Pennsylvania, Los Angeles and other American communities. Differential mortality and morbidity brings about genetic changes within populations, and micro-races offer the best opportunities at present for such comparative studies.

With respect to geographical races, local races and micro-races the question is not which is more important, but rather what questions we are trying to answer.

SUMMARY

The number of races of mankind which varies from no more than two to several hundred according to the taxonomy consulted, ceases to pose a major problem if the taxonomic category used is precisely defined.

Immediately below the species is the *geographical race,* a geographically-delimited collection of local races which may differ markedly, one from the other. *Local races,* in turn, correspond to natural or breeding populations, and are at once the units of evolutionary change and the common subjects for investigation. *Micro-races,* though not isolated geographically or

by extensive cultural prohibitions, still differ from each other in numerous ways.

Geographical races, local races and micro-races offer opportunities for very different investigations in relation to race. One is not more real or more fundamental than the other, but each provides the answer to different questions and the solution to different problems of ongoing evolution in man.

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III

RACE DIFFERENCES

Races differ in a great many respects—in language, in dress, in gesture, in body size and proportions, in amino-acid excretion patterns, in tooth morphology and in the proportions (or frequencies) of the various blood groups.

Those differences that are primarily learned, such as language or religion, may play an important role in maintaining the genetic isolates we call races. Languages are effective isolating mechanisms, as are religions. In various parts of the world there are *sympatric races,* occupying the same territory, but reproductively isolated because of religious practices or language differences. Yet, such learned differences in behavior are not biologically inherited: they may be concommitants of race, but are not a part of race.

Besides such learned differences between races, clearly nongenic in nature, there are many differences in body size and form that are responsive to environmental alterations. Stature is a type example. Though stature is in part inherited, nutritional adequacy during growth affects stature to a very large degree. Head-form, once considered a purely inherited trait has since been included among the "plastic" human features, susceptible to nutritional modification or to the effects of cradling and other skull-deforming practices. Such plastic traits, though showing considerable differences from population to population, are of questionable utility in racial comparison except when great caution has been used in untangling the effects of nurture from genetic nature.

The ideal traits for use in racial comparisons, and in the analysis of ongoing evolution within races, are those that are simply inherited and of known mode of inheritance. With such traits it is possible to go beyond *trait frequencies* (the proportion of indi-

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viduals showing the traits in question) to *gene frequencies* (the proportion of the allelic genes in question).* Gene frequencies, though mathematically derived from trait frequencies make for more accurate comparisons and facilitate calculations. That is why the blood groups, the haptoglobins, taste-blindness and the abnormal hemoglobins, finger prints and rare hereditary diseases are so useful in the study of race: knowing the mode of inheritance, phenotype (or trait) frequencies can be supplanted by gene frequencies.

Nevertheless, there are many racial differences, not simply inherited, but due to the cumulative effect of several genes, that are of marked utility in population comparisons. Such "polygenic" traits include hair form and skin pigmentation (excepting albinism), tooth form, and the extremes of body build. Not knowing the mode of inheritance, gene frequencies cannot be calculated and the mathematics of population comparisons is therefore hampered.

A further caution must be added about purely phenotypic differences, even if clearly inheritable. A given phenotype may be due to one set of genes in one population, and another set of genes in a second population. It is by no means certain that the genes for dark skin are the same in all populations and therefore the assumption of genetic affinity between the dark-skinned peoples of Africa and Melanesia is questionable. This caution applies with even greater force to such complex phenotypes as nose form or the extremes of stature. It is questionable whether the various "pygmies" of the world are related simply because they are pygmy, and it is a very tenuous assumption that the Ainu are related to Europeans simply because of their generalized hirsutism.

Thus it is that the neatly and simply inherited differences are currently of maximum use in the study of race, while other obviously inheritable but polygenic traits are of secondary utility until more is known about their genetics, and the appropriate mathematical methods have been developed.

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* Individuals of blood type O are homozygous for the gene, being of the genotype OO. But the majority of individuals of blood type A or B are of the genetype OA or OB. The gene frequency for O is therefore greater than the trait frequency. In the case of Mendelian dominants the situation is reversed. For computational methods see Boyd (1950) appendix A.

PIGMENTATION AND RACE

Nevertheless, racial differences in skin pigmentation deserve first mention, if only because such differences have been long observed. Among the lightest-skinned individuals of northern Europe there is little of the brown-black pigment called *melanin* in the lower layers of the skin, and the apparent color of the skin is largely due to reflections from the skin surface, with some of the blood pigments showing through. So-called "whites," of course are not white at all, but a light pinkish brown, reflecting from less than 20% of the light (in the blue end of the spectrum) to nearly 40% in the fairest. The "browns" and "blacks" have increasing amounts of melanin and in the darkest skins, no more than 1% of the light may be reflected from the unexposed skin areas.

To the best of our knowledge, skin pigmentation is primarily dark melanin in various amounts and in various degrees of dispersion. There are also yellow and yellow-red *pheomelanins,* but not known for man except perhaps in red hair follicles. At any rate there is no true yellow-skinned race and we need not invent a complicated ancestry for the Bushmen who have been described as having "yellowish" skins.

The amount of pigment in the unexposed skin, of course, is only half of the story, for the capacity to tan is also important, and this capacity is not invariably related to the unexposed skin color. Very little has been done on this tanning capacity but it figures large in racial appearances. A well-tanned Norwegian or Swede may be darker than an indoor-working American Negro. The light skins of aristocratic Polynesians or Arabs, need not be indicative of genetic differences but merely differential exposure to sunlight. On the other hand, the capacity to tan may be limited in even brunette-skinned individuals. Therefore racial comparisons must be limited to areas of skin ordinarily unexposed to the sun's rays or made in connection with controlled tanning studies.

THE HAIR

Next to the skin and its pigmentation, hair form, color and abundance have been most often utilized in racial taxonomies. In fact, hair form alone would effectively discriminate two-thirds of the world's populations.

The range of hair forms is wide, and in this respect, man is more variable than any other primate, though in all fairness no other primate occupies so wide a geographical range! In much of Asia and in aboriginal America, the head hair is straight or nearly so and coarse(over 100 microns). In much of Africa head hair is highly curved, even to the tight centimeter-wide spirals best seen in the Bushman and Hottentot. In Europe there is a wide range of hair forms, from nearly straight—but rarely of Mongoloid coarseness—to helical, as seen in Greek statues of Pericles' time. A world survey of head-hair form is given below.

WORLD DISTRIBUTION OF HAIR FORMS
(By Geographical Areas)

Geographical Area	Straight Coarse	Straight to Wavy	Helical to "Woolly"	Spiral Tuft
North and South America	X	X		
Polynesia	X	X	X	
Australia		X	?	
Papua—New Guinea		X	X	
Asia	X	X		
India		X	X	(?)
Africa		X	X	X
Europe		X	X	

Hair on the body, less extensively studied, is characterized by great racial variability. In most of Asia, America and much of Africa, body hair is sparse or absent. In Europe, and the Middle East to Afghanistan and Pakistan, body hair is often well-developed. Sporadically, generalized hirsutism exists—in Papua and New Guinea, among the Australian aboriginals, and in the Ainu of northern Japan. However, the hairiness of the Ainu has been exaggerated as a brief stay at any American bathing beach will confirm.

Balding, to be more exact, male pattern balding constitutes a very real racial difference. Hormone-mediated and gene-de-

termined, it is rare among Asiatics, Amerindians and Africans, and common in Europe and the Middle East. Balding is associated with, but genetically independent of generalized hirsutism. At its extreme manifestation, male pattern balding begins in the early twenties. Possibly, if Italian data hold for other countries, early male pattern balding is associated with increased fertility.

THE BONES

Racial differences in the size, proportions, form and mineral content of the bones are well documented for a number of groups, since these variations have been extensively studied on skeletons.

Using trunk-length as a reference, the relative proportions of the limb bones vary markedly from Eskimo and Japanese, at one extreme, to American Negroes and especially certain African groups at the other extreme. Racial differences in the relative lengths of the metatarsal bones of the feet as expressed by differing digital formulae are obvious even in the living, as are racial difference in the calcaneus and astragalus.

The presence of accessory or "Wormian" bones along the suture lines of the skull, typically between the occipital and parietal bones where they meet at the back of the head, is characteristic of Asiatics and especially American Indians (Fig. 24). This alone is a great help to law-enforcement officers, when presented with an exhumed skeleton to identify. Accessory or suture bones distinguish recent white burials from older Amerindian remains.

Medically, Negro-white differences in the volume of the sacral canal become of importance when certain types of spinal anasthesia are contemplated. Racial differences in the architecture of the pelvic bones are often so marked as to make identification possible from the pelvis alone.

Recent studies on bone density reveal marked differences between American Negroes and American whites. The weight/volume ratio is higher in Negro skeletons indicating a greater degree of mineralization. This difference is in the opposite direction of what might be expected on a purely nutritional basis.

THE DENTITION

Many racial differences in the dentition are known and documented, due in part to the fact that the teeth are equally accessible to study in the living and in skeletal collections as well.

Tooth size varies considerably from population to population, the largest teeth being reported for Australian aborigines, and the smallest for the European Lapps. In the former, the crown length of the first molar averages 13.0 mm. (in the male) as compared with slightly less than 11 mm. in the Lapps. Notably, there are marked differences in tooth size within each geographical race. For example, there are both small-toothed and large-toothed local races in Africa and the teeth of Greenland Eskimos are larger than those of the Aleut.

The form of the teeth also differs from group to group (Fig. 6).
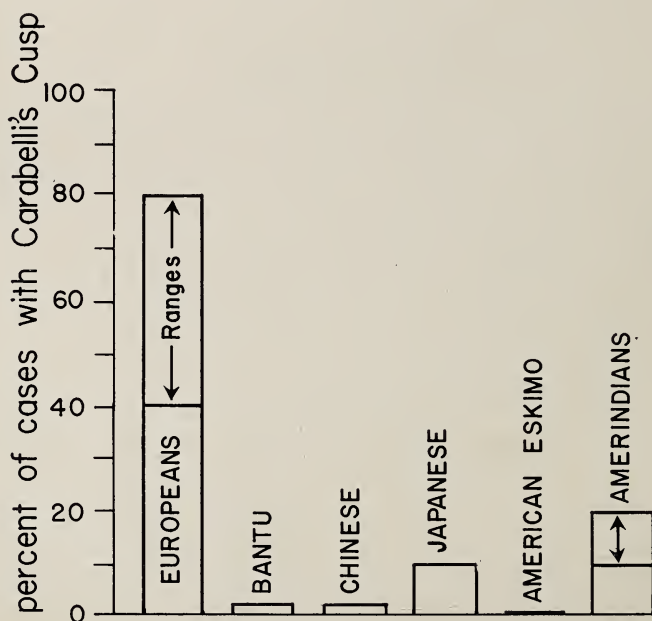
Fig. 6. Frequency of Carabelli's (accessory) molar cusp in different groups showing maximum frequencies in Europe. Where frequencies vary the ranges are indicated. (Data from Carbonel, V. M.: The tubercle of Carabelli in the Kish dentition. *J. Dent. Res.*, 39:124-128, 1960.)

Among many Asiatic populations the third molar roots are fused, whereas this condition is less common in Europeans and least so in many Africans. The number of cusps on the posterior teeth is similarly variable, with cusp reduction evident in both European and Asiatic peoples. The most impressive racial variation in the form of the teeth, the "shovel-shaped" trait, is especially well developed in some American Indian tribes. Whereas the back surface of the incisors is slightly "shovel-shaped" in about 15% of Finns, the incisors may be rolled in almost tubular shape in Pima, Navaho, Hopi and other Indians, in whom the shovel shape may extend to the canines as well (cf. Fig. 7).

Finally, agenesis (congenital absence) of the third molars, rare in most Africans and verified in perhaps 6% of Europeans, may reach 30% in some Amerindian, Eskimo and Asiatic groups.

GROWTH AND RACE

Since the rate of growth and the timing of maturation are both greatly affected by the caloric reserve, few definite statements about racial differences in growth progress can now be made. Popular notions often turn out to be wrong, as for example, the supposition that children from southern or darker-skinned races mature earlier.

Nevertheless, some racial differences in growth are clear-cut, among them differences in the absolute and relative growth rates of the limb bones in American Negroes, Japanese, and American "whites." Here, the characteristic differences in leg proportions are well established during fetal life, as Adolph Schultz (1926) has shown.

Another racial growth difference involves the age at calcification of some of the bony nuclei of the wrist, foot and leg which appears to be earlier in American Negroes and in selected African populations. This may represent a simple difference of patterning, or it may be associated with osseous and neuro-muscular advancement during infancy as Dean has demonstrated.

For the teeth, however, the data appear to be incontestable. Almost every group averages earlier in the eruption of the permanent dentition than do Europeans or Americans of European origin. Even with the disadvantages of malnutrition, disease or

A

B
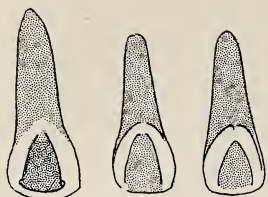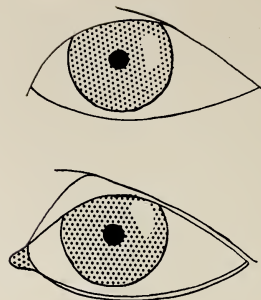
NEGROES
xxxx AMERICAN INDIANS
::::::: ASIATICS
//// WESTERN EUROPEANS
\\\\\ MIDDLE EASTERN
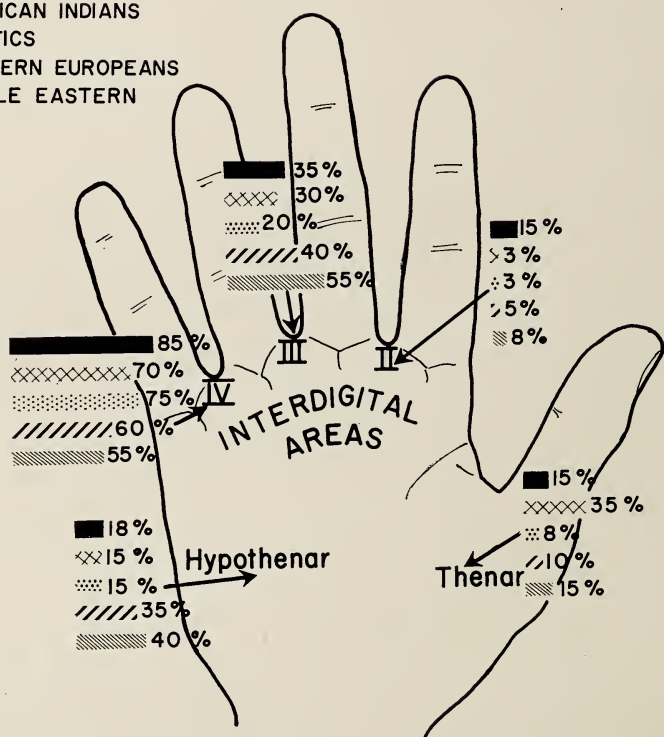
Fig. 7. Three areas of racial differentiation. A, the shovel-shaped trait on the posterior aspect of the incisors, especially common in the American Southwest. B, the inner or "Mongoloid" eyefold as contrasted with a typical European eye. C, geographical differences in specific palm-crease pattern frequencies documenting Amerindian-Asiatic differences in the thenar-first interdigital and the third interdigital areas. (From Rife, D.: Dermato-glyphics as ethnic criteria, *Am. J. Hum. Genet.*, 6:319-327, 1954.)

capture and life in concentration camps, Navaho, Maya Indian, Formosan, American Negro and Aleut children are ahead of norms for British and American "whites." Third molar eruption, rare before age 17-20 in most Americans, has been reported as early as the twelfth year in Kenya natives. These differences in tooth eruption are probably paralleled by racial differences in tooth calcification, a suggestion supported by a preliminary analysis of one American Indian group.

PHYSIOLOGICAL AND BIOCHEMICAL DIFFERENCES

Investigations in human physiology and biochemistry have revealed a great many apparent racial differences, but in most cases the purely genetic nature of these differences have as yet not been confirmed. Thus, most "natives" have lower blood pressures and lower serum cholesterol levels than is true for individuals of western European origin, but both blood pressures and cholesterol levels rise with urbanization and westernization. "Racial" differences in the basal metabolic rate after correction for temperature appear to be largely functions of body build. Much more convincing are differences in drug-sensitivity (chapter VII), abnormal haemoglobins and blood groups discussed in later chapters, and the haptoglobins to be mentioned shortly.

Differences in the amount of excreted 17-ketosteroids do seem to be well-documented with considerably lower excretion levels in South African natives, as measured both in Africa and England, citing the work of Barnicot and Wolffson (1952). This does not necessarily mean that Africans are lower in the production of androgenic steroid hormones. It may mean that, through differences in either adrenal-cortical activity or liver metabolism they excrete less of the C-17 steroids and more of the C-20 steroids not measured by the reagent used. Male pattern balding may also be considered along with 17-ketosteroid excretion levels, but as an example of hormone target-organ relationships. Since American Indians do not develop male pattern balding following testosterone administration, their resistance to balding may be attributed to target-organ insensitivity rather than to hormonal insufficiency (cf. Hamilton, 1951).

Best documented to date, of the apparent racial differences in

urinary excretion patterns, is the excretion of beta-amino isobu-
tyric acid (BAIB) in Japanese, Chinese and Amerindians. Rarely
excreted in adult Europeans, this is a common excretion product
in Orientals tested both in Asia and in the United States. BAIB
excretion is also found in Chinese and Japanese in America sub-
sisting on purely occidental foods (see Fig. 8). So far BAIB holds
the same position among the biochemical traits that the Diego
factor holds among the blood groups, straight, coarse hair among
the hirsute traits, and the suture or Wormian bones hold among
the osseous differences. All are suggestive of "Asiatic" origins.

THE HAPTOGLOBINS

In addition to marked racial differences in the frequencies of
the various blood group alleles (Chapter IV), and in the fre-
quency of the "abnormal" haemoglobins (Chapter VI), there is
increasing evidence as to the differential distribution of the gene-
determined haptoglobin "types."

The haptoglobins, haemoglobin-binding proteins, are stable
proteins, well differentiated by electrophoretic analysis of blood
sera. There are at least three haptoglobin types Hp 1-1, Hp 2-1
and Hp 2-2 (Fig. 9) apparently controlled by the genes Hp^1 and
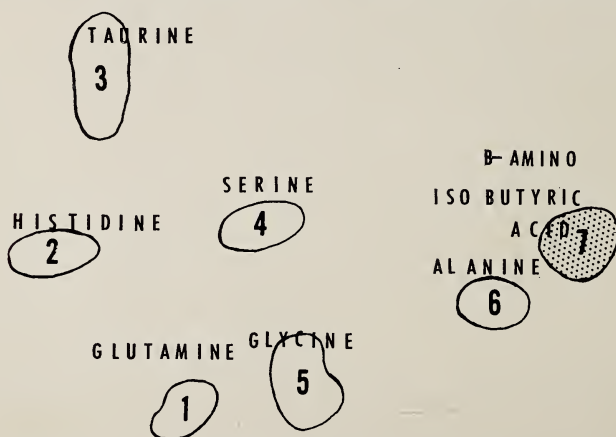
Fig. 8. Diagram of a urinary chromatogram showing the area occupied by
amino acids including BAIB (β-amino isobutyric acid). This amino acid
is rarely excreted by Europeans and Africans, but frequently by Asiatics and
Micronesians. Compare with frontispiece and see text.

Hp². A person heterozygous for Hp¹ and Hp² therefore exhibits the haptoglobin type Hp 2-1.

The Hp¹ gene, the less common allele in Europe, has a frequency of about 0.36 in Finns, Norwegians and Basques. It is more common in American Negroes (gene frequency approximately 0.41). As might be expected, therefore, the Hp¹ gene is the predominant haptoglobin allele in some parts of Africa with a gene frequency of 0.59 in Nigeria and in excess of 0.73 in Liberia. The Hp¹ gene is far lower in the Bushmen, according to a recent report.*

TASTE-BLINDNESS AND RACE

Racial differences in dietary practices have long been viewed with interest and the suggestion has been made that people who prefer hot, spicy foods differ in taste-acuity from those who

Group	Starch gel	Starch block	Ultracentrifugal pattern
1-1			
2-1			
2-2			
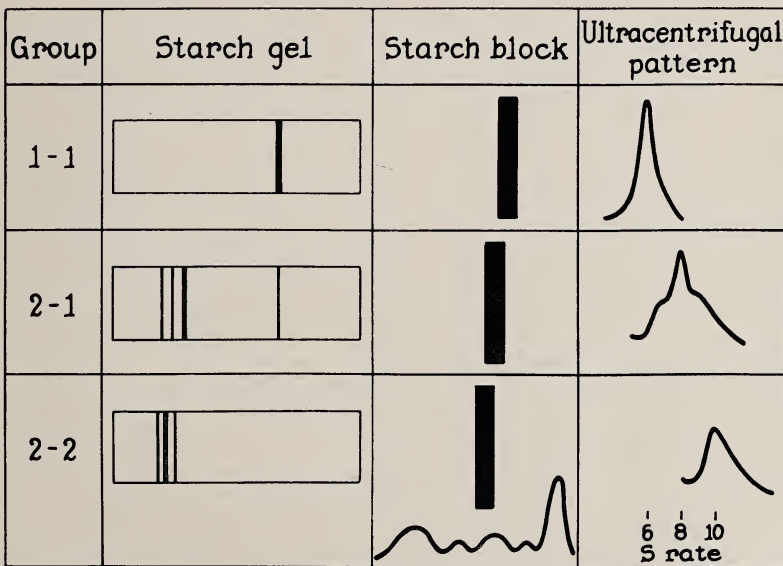
Fig. 9. The three haptoglobin phenotypes Hp 1-1, Hp 2-1 and Hp 2-2 as shown by starch-gel electrophoresis, starch block electrophoresis and by the centrifugation of blood serum. (From Bearn, A. G., and Franklin, E. C.: Some genetical implications of physical studies of human haptoglobins, *Science, 110*:596-597, 1958.)

* Barnicot, *et al.: Nature, 184:* 2042, 1959. Both in the Americas and the New World the frequency of the Hp-1 phenotype increases toward the Equator.

espouse the cult of culinary blandness. So far, however, conclusive evidence as to racial differences in the classic taste parameters (sweet, sour, salty and bitter) has not been achieved though there are gene-determined differences in sensitivity.

Nevertheless, there are individual differences in the capacity to taste *phenylthiocarbamide* (PTC) and a large number of related substances, all of which have some antithyroid activity. These differences are apparently controlled by a pair of allelic genes T (for tasting) and t (non-tasting): the genotypes TT and Tt are tasters, and the tt genotype corresponds to the non-taster phenotype. However, there is an hormonal effect as well, as evidenced by the greater proportion of taste-sensitive women. From the work of Fischer and Griffin (1959) taste-sensitivity may be explained by the genetically-determined level of di-iodo tyrosine in the saliva.

Taste sensitivity varies from population to population. Among American Indians the majority are tasters. In fact some Amerindian populations must approach 1.00 for the taster (T) gene. In Africa and the Middle East the tasters are in the majority. However, in numerous parts of Europe the gene frequency for T is considerably lower and there are up to 43% of non-tasters in India (Fig. 10).

Boyd has suggested that tasting is adaptive in some areas of the world, and inadaptive in others. At the present time the world distribution of PTC-propylthiouracil tasting not only evidences marked race differences but suggests many experiments bearing on PTC-taste sensitivity. "Tasters" for example, probably have more food aversions than non-tasters, a quality of limited value in times of scarcity.

RACE DIFFERENCES

Race differences exist throughout the body and into the area of metabolic activity and biochemical functioning. Not only are there race differences in the pigmentation of the skin, eyes and hair, and in the morphology of the lips, nose, eyelids and mouth, but there are also differences in the inner organs, in the muscles and in the patterning of subcutaneous fat. The teeth, hemoglobins and hemoglobin-fixing proteins, taste acuity, drug sensitivity, urinary excretion patterns and probably sex-hormone activity
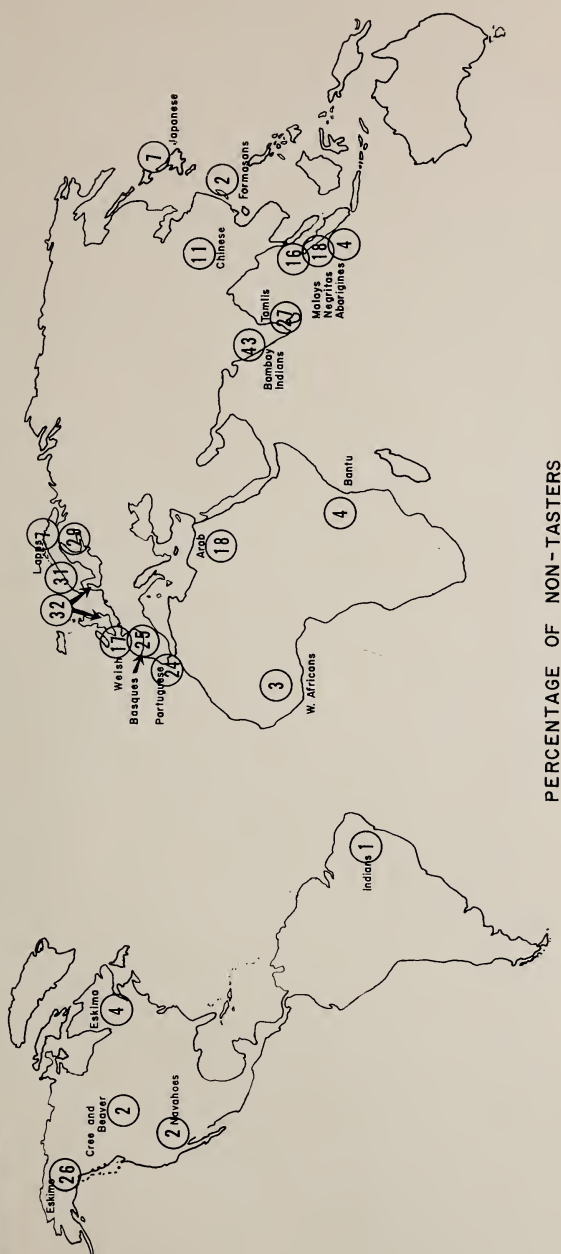
PERCENTAGE OF NON-TASTERS

Fig. 10. Percentage of individuals recorded as "non-tasters" of PTC (phenylthiocarbamide) in different geographical areas. Note the high proportion of non-tasters in India, Europe and among the Alaskan Eskimos. (From Allison, A. C., and Blumberg, B. S. Ability to taste phenylthiocarbamide among Alaskan Eskimos and other populations, *Human Biol.*, 31:352-359, 1959.)

exhibit measurable differences in averages or proportions from race-population to race-population. To say that "race is only skin deep," a statement notably but inaccurately repeated in many textbooks, is patently naive in the extreme.

Some of the race differences are so marked as to allow little overlapping between geographical races. By way of example, the extreme spiral-tuft form of the head hair is virtually unknown in Europeans. The Rh negative gene, on the other hand, is rare outside of Europe. For many other traits, it is the frequencies or proportions that are distinctive, as with the haptoglobin types in Europe and Africa. Often, differences are of a small order of magnitude, large enough to attain statistical significance but not impressive enough to use in a taxonomy.

Not infrequently, particular differences *within* geographical races are more marked than those between geographical races. For example, the phenomenon of steatopygia (fat rears) virtually sets off the Bushmen-Hottentot from the rest of Africa, and in body build the Papago are distinct from other Amerindian groups. But a single such difference probably reflecting local selective forces does not alarm the taxonomist, no more than (and for the same reasons) that a run on the bank does not affect the long-term position of a gambling casino.

It is important, however, to emphasize the independence of genes. The fact that genes P and Q are both common in a given group does not mean that they are inherited together, that they came from the same source. Whereas the PQ genotpye may be characteristic of a given population, individuals who are pp or qq are not less "pure" on that account. And since the genes are independent, the existence of the gene P in a given population need not be evidence that it came from a PQ source. In other words, dark skins are not necessarily of "Negro" origin, nor internal eye-folds inevitably due to "Mongoloid" ancestry. Failure to recognize the independence of genes has led to some rather implausible historical reconstructions in the past.

Races differ in a great many gene-determined respects and a marked difference in the proportion of one set of alleles predicts nothing in respect to the proportions of another set. Moreover, it is not the mere fact of difference that intrigues us today, but

rather the source of the difference. The fact that one race has a high frequency of some trait, and there is a low frequency in another race, is mildly interesting as a descriptive fact but no more until we turn our attention to the reasons why.

SUMMARY

Racial differences are known to exist in almost every area of anatomy where comparative data have been accumulated, and there is growing evidence for race differences in biochemical functioning and in the constituents of cells and tissues.

In a minority of examples, there may be little overlapping between geographical races. Much more frequently, differences are merely matters of proportion, the incidence of different traits, or the frequencies of the allelic genes that determine them. Not infrequently a given gene-determined trait varies more extensively among local races in the same geographical race than between geographical races.

The independence of the genes that make for similarities in differences must be clearly understood. Thus two races may be markedly different with respect to one allelic pair of genes and not at all different with respect to another. Taken alone, therefore, a particular set of differences (or similarities) says nothing as to communality or divergence of origin or descent.

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IV

BLOOD GROUPS AND RACE

For many centuries the possibility of transfusing blood has appealed to surgeons. Great numbers of men died from loss of blood following accidents or battle. Patients expired during and following operations, patients who could have been saved by blood transfusion. But until 1900 transfusions were impractical: too often they ended in shock and death. Somehow, blood just didn't mix.

Then, about fifty years ago, Moss and Jansky discovered the existence of four different blood types which they named types I, II, III and IV. By determining the blood "type" of the patient, and transfusing compatible blood, blood transfusions became a fortunate practicality. And, as a result of the extensive work on blood-typing, during and since World War I, a tremendous amount has been learned about blood types and their relation to local and geographical races.

THE ABO SYSTEM

The original four types of Moss and Jansky have since been renamed A, B, AB and O. These blood types are simply inherited with both A and B dominant over O. Genetically, type O blood is OO, while the phenotypes A and B may be either AA or OA, and BB or OB respectively.

Blood Type	Possible Genotypes
A	AA, OA
B	BB, OB
AB	AB
O	OO

Of the three blood types (or factors) in the A-B-O system, O is the most common the world over. Among some American

Indian groups, over 90% of the people are of blood group O, with gene frequencies for O as high as 0.99. In much of Europe, the incidence of O is from 35% to 40%, and the frequency of the gene r is between 0.6 and 0.7. Among Chinese, Japanese and Asiatic Indians and in many African populations, O may be as low as 30% with a gene frequency of as little as 0.5. However, even within a particular geographical race there are marked differences in the incidence of O. By way of example, O has been reported in 97% of Utes, but in only 23% of Blackfeet Indians. In Europe there is an increasing incidence of blood type O from south to north, a trend that is repeated even within the British Isles.

Blood group A, the next most frequent blood type the world around, similarly evidences its own pattern of distribution. It is rare (under 5%) in some Amerindian populations, yet extremely common (over 75%) in others; both are world extremes, and point to the wide divergences possible between local races. By way of comparison, the phenotype frequency of A is about 45% in England (with a gene frequency of 0.25), and the same among Americans of Northwest European descent.

Blood group B, the least common of the three types in the A-B-O system, has the most interesting world distribution. It is completely absent in most North and South American Indians, rarely over 2% in others, and this may be due to admixture. Blood group B is less common than A in Europe, with a phenotype frequency of 9-25%, averaging about 15%. However, B which increases in frequency to 22% in Ukrainians and 25% in Egyptians, reaches maxima of 35-37% in China, Java, Bengal and the Siberian north. B is an Asiatic and African blood type much more than it is European and it is not at all Amerindian.

Comparing Sumatra, Java and the Philippines with Polynesians, there is a great divergence in A-O-B blood groups. The Asiatic-Malaysian areas are high in B, whereas Polynesia is low in B (under 3%). Clearly Polynesians cannot be derived from any recent mixture with Malaysian people. In similar fashion, the low to zero incidence of blood group B in aboriginal America precludes major recent contact with Asiatic Mongoloids whose frequency of B ranges from 20 to 40% in different populations.

However, blood type A is divisible into two subtypes, A₁ and

A_2; of the two A_1 is far more common than A_2. The incidence of A_2 varies from zero (in Amerindians and Australians) to 10-15% in much of Europe. American Indians agree with Asiatic Mongoloids in the virtual absence of A_2, as is true of Polynesia and Australia as well. In fact, A_2 is practically limited to Europe and Africa.

Broadly, the A-B-O blood system can be summarized by noting (1) the near absence of B and absence of A_2 in the Americas, (2) the low to moderate frequencies of B and increasing frequency of A_2 in Europe, and (3) the high incidence of B and relative rarity of A_2 in Asia. But the Australian aborigines seem practically Amerindian, as do the Polynesians in their low incidence of B and absence of A_2. The A-B-O system, taken alone would suggest a separate origin for them, whereas it groups Asia, India and Africa.

THE MNS-U SYSTEM

Next in order of discovery to the A-B-O system, and therefore in the amount of information we now have, is the MNS-U system. For years it was the M-N system, then S was added, and more recently U (sometimes written S^u).

As with A and B, M and N are inherited without dominance, A person may be M, N or MN, corresponding to the genotypes MM, NN and MN. For much of the world, the frequencies of M and N are about equal, and rarely is either M or N entirely missing. Thus, in England the gene frequencies for M and N are close to 0.53 and 0.47 and in Japan they approximate 0.56 and 0.44 respectively.

But M is peculiarly high among the American Indians, and in many Amerindian tribes there is little or no N. (Low values for N have also been reported for the Near East). In contrast to the situation in America, Australia is the virtual homeland of N, and M—that is MM individuals are entirely absent in some aboriginal hordes. Throughout the Pacific, in Papua, Fiji and Hawaii blood type N is similarly predominant over M. Whereas these peoples of the Pacific are similar to Amerindians in the low incidence of B and of the subtype A_2, the high N frequencies of Pacific people set them off completely and preclude recent major contact with America, as their absence of B does with Asia.

Since the extremes of M and N are found in populations formerly explained on the basis of "admixture" it is notable that no combination of Asiatics could yield the low N values common in America, and no Caucasoid-Negroid combinations could yield the nearly M-free peoples of Australia. Either the "original" three races never existed, or subsequent evolution has so altered their genetic makeup in particular localities as to make proof of the three-race hypothesis quite impossible.

Still, two additions to the M-N system must be made. One involves the rare gene S, discovered in 1947, which involves a mutation alternately from M or from N. A person may be MS, MNS or NS. The S gene, discovered in England, (where it is quite common) has since been shown to be absent in Australian aborigines, but present in natives of New Guinea. Tentatively, therefore, one may suggest either closer affinities between Papuans and Europeans, or an absence of ties between the Australian and European geographical race. Perhaps MS and NS might be called M_2 and N_2, in which case we could say that both M_2 and N_2 are fairly common in Europe, but absent among the aboriginal Australians.

The second addition to the MNS system involves the gene U, sometimes written as S^u. Among Europeans everyone is U-positive. In Negroes, or more specifically the American Colored, about 1% are U-negative as shown below. This polymorphism leads to complications, both in transfusions and in childbirth. Transfusing U-positive blood into a U-negative Negro can be dangerous. While segregated blood stores would not be the answer (most Negroes are U-positive as are nearly all Europeans) the necessity to consider race in planning transfusions becomes all the more apparent. Further divergences in Negro blood groups will be mentioned later in this chapter.

U-NEGATIVE PHENOTYPE AND GENE FREQUENCIES

	No.	U+	U−	Gene Frequency
Milwaukee "Caucasians"	10,000	10,000	0	0.00
Milwaukee Negroes	1,429	1,425	4	0.05
New York Negroes	989	977	12	0.11

From Greenwalt, Sasaki, Sanger and Race (1956) and Sampson and Thomas (1959).

RHESUS AND RACE

By now, most educated individuals have heard about the *Rhesus factor* and Rh incompatibility. They know that, especially in later pregnancies, a rhesus-positive fetus can be damaged by antibodies produced by the Rh-negative mother.

Actually, there is a long series of rhesus genes, R_1, R_2, R_0, r', r variously written as R', R'', etc. Practically, R_1, R_2, r' and others are dominant over the rhesus negative gene, which will therefore be written as r;* the rhesus-negative individual is a homozygous rr.

Among Europeans in whom the rhesus-negative gene was first discovered, the proportion of Rh negative individuals ranges from about 12% through an average of approximately 15% in England and the United States, to nearly 30% in Basques.

Elsewhere in the world, rhesus-negative blood is rare, uncommon or even totally absent. A long series of American Indians and many series of Papuans or Australian aborigines can be compiled with few or no rhesus-negative individuals. Rhesus-negative blood is also absent in Polynesia, and uncommon in China, Japan and the Philippines, though the incidence begins to rise in India, Pakistan and Afghanistan.

Among American Negroes the type rr is about half as common as in northwest Europeans, leading expectably to its relative rarity in the sections of Africa whence their African genes originated. However, the type R_0, which rarely exceeds 2% in Europe, achieves maxima of more than 40% in the American Colored, and over 70% in Africa. It is for this reason that the R_0 gene in Melanesia is of interest.

The present distribution of the "rhesus" blood types, the high frequency of the gene r in Europe and especially among the Basques, the rarity of r in Asia, Australia, Polynesia and aboriginal America, and the concentration of R_0 in Africa, cannot be explained in any simple way. One is loath to accept Boyd's "Early Europeans," who presumably contributed vast numbers of the

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* The notation for the Rh alleles used here is primarily that of Alexander Weiner though it does not follow his more recent revisions. Abroad there is a totally different notation developed by R. A. Fisher, which involves different assumptions about the mode of inheritance.

rhesus-negative genes in Europe and were then absorbed by late-comers. Even if there had been such a population, we are still faced with the continued survival of this disadvantageous gene, the more so under primitive conditions. And we wonder what value R_0 has in Africa.

DUFFY-AN AUSTRALASIAN GENE

In 1950, a new blood-group factor, unrelated to the A-B-O, MN and Rh systems, was found in the blood of a Mr. Duffy, and the new system that eventuated from this chance event was named after him. The Duffy factor soon proved important in transfusions, as a cause of transfusion reactions.

Briefly, there is the Duffy-positive gene (Fy^a) and the Duffy negative allelomorph (Fy^b). A person may be Fy^aFy^a, Fy^bFy^b or Fy^aFy^b. However, since Fy^a is dominant over Fy^b, only two phenotypes are identifiable. Therefore, phenotype frequencies of Fy^b are used in computing the gene frequencies for Fy^b and Fy^a respectively.

In England 65% of subjects proved to be Duffy-positive, corresponding to a gene frequency of 0.40. Far higher gene frequencies for Fy^a have appeared in Pakistan, India and among New York Chinese, and Australians. Far lower gene frequencies were observed in American Colored individuals (see below).

FREQUENCIES OF THE DUFFY-POSITIVE
(Fy^a)GENE

Group Studied	Gene Frequency Fy^a
Cape York Australians	1.00
Koreans	0.99
Chinese	0.91
Japanese	0.86
East Indians	0.73
American Indians	0.50-1.00
English donors	0.41
Minnesota Whites	0.40
American Colored	0.14

From Matson and Swanson (1959), Simons *et al.* (1958) and Race and Sanger (1954).

Obviously, the home of the Duffy-positive gene is in the Pacific and Eastern Asia with decreasing frequencies both southward into the Americas and westward into Europe and then Africa where the Fy^a gene is virtually absent. The distribution of Fy^a, if taken seriously by proponents of a simple three-race theory, would provide better evidence that the Europeans are of mixed Polynesian-Negro origin (since they are intermediate in Fy^a frequencies) than for any tri-hybrid origin for the Polynesian peoples!

DIEGO, AN "ASIATIC" BLOOD GROUP

The Diego blood group system, one of the most recently discovered, involves a pair of genes Di^a and Di^b, and the two phenotypes, Diego "positive" and Diego negative. Though Diego-positive individuals are nowhere in the majority, the Di^a gene clearly separates Australia and the Pacific from Asia and the Americas (Fig. 11).

In two series of Australians, one in the north nearest New Guinea, and one in Central Australia, no Diego-positive individuals were found. Similarly the Diego antigen appears to be absent in the eastern Polynesians. On the other hand, Diego-positive individuals have been reported in 25% of Peruvian Indians and 10% of Penobscot Indians, but rarely among Alaskan (Tlingit) Indians and Alaskan Eskimos. Several studies confirm the virtual absence of Di^a in Alaska, and the relatively high frequencies in Central America.

Despite the absence of the dominant Di^a gene in Alaska, Diego-positive individuals are frequently found among Chinese, Japanese and Koreans. This suggests, quite reasonably, an Asiatic source of Diego in the Americas. However the extremely variable phenotypic frequencies in Central and North America, ranging from 0% to over 20% and the absence of Diego nearest the Asiatic mainland can be explained only in terms of local selection.
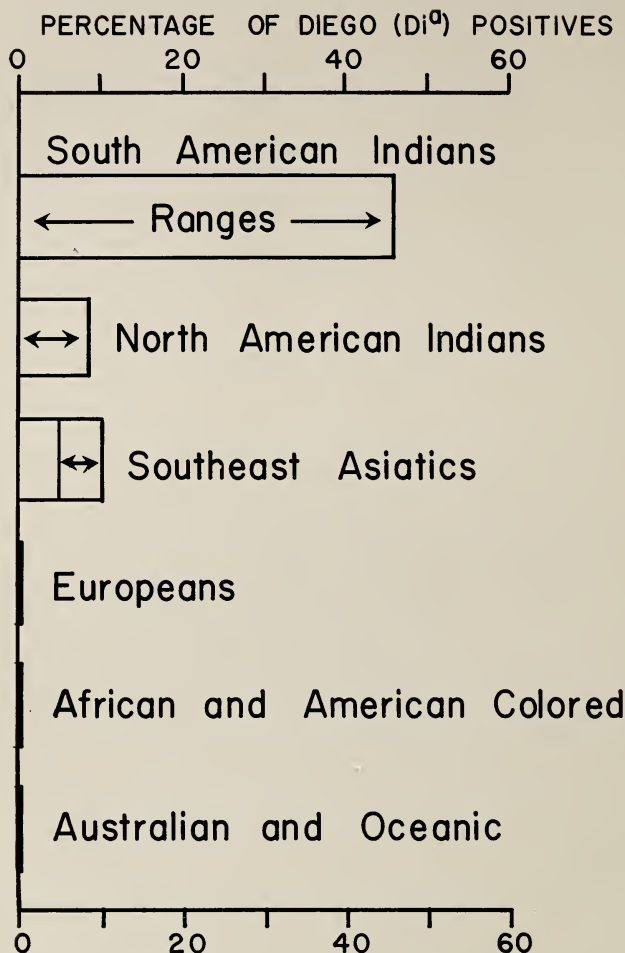
Fig. 11. Percentage of Diego-"positive" individuals in different populations. The genotype Di^a or Di^{a+} is largely restricted to Amerindians and Asiatics. (Adapted from Layrisse, M., Wilbert, J., and Arends, T.: Frequency of blood group antigens in the descendants of Guayqueri Indians, *Am. J. Phys. Anthropol.*, N.S. *16*:307-318, 1958.)

SUMMARY OF PRINCIPAL BLOOD GROUP SYSTEMS

Blood Group System	*Description of Phenotype Frequencies*
A-B-O (including A_1 & A_2	O most common group, with over 50% of individuals in most populations of this type. B nearly absent in Aboriginal America and Australia, progressively more common in Europe (15%), Africa, India and Asia (up to 40%). A_2 practically limited to Europe.
MNS-U (or S^u)	American Indians almost exclusively M, N most common in Australia and the Pacific. MS and NS absent in Australia. U-negative rare, apparently limited to Africa.*
Rh (R_1 R_2 R_0 r' r etc.)	Rh negative individuals (rh) rare or absent in most of the world, but approximate 15% in Europeans. Of the Rh positive alleles (R_1, R_2 etc.) the R_0 form is primarily found in Africa (up to 70%).
Duffy (Fy^a Fy^b Fy)	Most Australians and Polynesians§ and 90-99% of Asiatics Duffy-positive (Fy^a), 90% in India, 85-90% of most American Indian,** 65% in England and America, 27% in American Negroes. Fy^a very low in Africa but the gene Fy extremely common (>80%).†
Diego (Di^a Di^b	Diego-positive (Di^a) individuals limited to Amerindians (2-20%), and Asiatics.* Di^a absent in Europe and Africa, Australia, Micronesia and Polynesia,§ and in Eskimos.*
Kidd (Jk^a Jk^b)	Kidd positive (Jk^a) most common in West Africa and American Colored (>90%), North American Indians (70%-90%), Europeans (approximately 70%), and least common in Chinese (50%-55%).† **

Special References: * Greenwalt *et al.* (1958), † Race and Sanger (1954), § Simmons, Graydon and Gajdusek (1958), ** Matson and Swanson (1959).*

BLOOD GROUPS AND NATURAL SELECTION

As a result of numerous investigations involving millions of people, it is extremely clear that the blood groups are subject to natural selection. Population differences in blood group frequencies may therefore be viewed as the product of competing lines of selection, the balancing of selective advantages and disadvantages associated with each serological type.

Classic Rh incompatibility, the loss of a Rh heterozygous infant

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* Levine has shown a protective effect of certain ABO genotypes against Rh incompatibility. See *Human Biol.*, 30:14-28, 1958.

due to antibody formation in a Rh-negative mother, is the best known example of selection against a blood type. The long-term effect of such incompatibility would be to eliminate the Rh⁻ gene (r) from the population, and this may explain the rarity or absence of the Rh negative gene in much of the world. At the same time we need some explanation for the continuance of this gene in Europe, especially among the Basques.

Less well known, but equally important, is maternal-fetal incompatibility in the A-O-B system. Since the net effect would be to eliminate types A and B differentially in comparison to O, this may provide an explanation for the numerical predominance of O the world around. Moreover, now that transfusion reactions have been discovered for a variety of other blood group factors, including the rare U in the MNS-U system it is obvious that the increasing use of blood transfusions is serving to eliminate some proportion of the less common genes in each population.

Apparently, serological incompatibilities are one cause of infertility. The A-B-O phenotype frequencies proved markedly abnormal in a series of infertile couples studied in Michigan. Since such incompatibilities are not likely to occur in O couples, there is obviously a slight adaptive advantage to the OO genotype. Reproductively, at least, the type O male is a "universal donor."

Recently, associations between A-B-O types and chronic illnesses have been demonstrated, among them an association between type O and ulcers and type A and gastric cancer. Although criticizied on statistical grounds and challenged outright by Weiner, similar results have been obtained in Europe, America and Japan. However, since the diseases involved occur late, commonly after the reproductive period, the importance of this kind of selection remains open for study.

In view of the fact that the blood groups are subject to selection the use of serological data in historical reconstructions is obviously limited. It would be unwise to guess as to the frequency of B in Asia ten thousand years ago, or even the frequency of the Rh negative gene among "early" Europeans. However, since all lines of selection now known would tend toward population homozygosity, special attention should be given to situations where, as with the Duffy factor in Europe, maximum number of heterozy-

gotes exist. There may well be a real adaptive advantage to being MN, Fy^aFy^b, and possibly AB. In fact, there are more MN children from MN x MN marriages than one might expect even if technical errors are to some extent involved.

BLOOD GROUPS AND HUMAN TAXONOMY

The blood groups are simply inherited and therefore qualify as ideal traits for use in racial comparisons. Blood group determinations can be made with relatively high reliability except for the less common antigens (Osborne, 1958), and this commends them for general use. Besides, blood group determinations are made by the millions every year in the course of blood donating and transfusions, thus providing a wealth of free information on blood groups in different races.

All of these advantages were clearly recognized over thirty years ago, and the idea of a serological taxonomy was advanced. After all, blood groups are genetical, accurate, stable and susceptible to statistical analyses. A serological taxonomy made at least as much sense, and in fact more, than a classification based on hair form or skin color.

But the first serological taxonomies were hardly reasonable. Using the A-B-O system, Asia, India and Africa ended up in one pile (based on the frequency of B), and aboriginal America and Australia in a second sorting (based on an absence of B). Totally unrelated populations were characterized by similarities in A-B-O frequencies and contiguous groups compared serologically were practically in separate planets.

The situation improved somewhat with the discovery of the subtypes of A, which belatedly confirmed Amerindian-Asiatic similarities and no longer made Europe an apparent Asiatic-Australoid mixture. M-N data further appeared reasonable, geographically as well as genetically. The most unusual distributions of M and N were geographically most isolated from Europe, Asia and Africa.

With the discovery of the Rhesus system of alleles, serological and geographical taxonomies became more nearly reconciled. Africa appeared clearly separable from Asia (despite high frequencies of B) and Europe from both. Even so, considerable

juggling was necessary to make serological taxonomies coincide with natural populations. What had happened to the serologists was exactly what had happened to morphologists. Using the conventional systems of differences they had confused their criteria (here the blood groups) with the races they were trying to describe. By near-sightedly working with the gene frequencies and ratios, they had come up with an artificial classification, a system of blood groups rather than a classification based on natural populations. To the extent that serological classifications worked, they were attempts to describe natural populations (as Boyd has done in his Boston University Lecture).* When the classifications were based on serological criteria, rather than the populations, the results were understandably bizarre.

Actually, the major contribution of the blood groups is not the establishment of a taxonomy, but to the more adequate comparison of related race-populations, their similarities and differences. By way of example, the virtual absence of B in Australia (except in the Cape York area) and the absence of S (that is MS and NS) confirms the separation of Australian and Papuan peoples. At the same time, the absence of Diego and the high frequencies of N, align the Australians with other peoples of the Pacific. Similarly, the similarities between Amerindians and Asiatics in the Duffy factor and in Diego, are balanced by the differences (in blood group B and possibly in the Kidd blood group).

Particularly for populations formed by recent admixture, the blood groups afford precise quantification. Using the Rh-negative gene (r), the R_0 gene, the incidence of the U-negative trait, Duffy (that is Fy^a) and the Kidd-positive trait, very exact estimates of the degree of intermixture can be obtained for the American Colored and Cape Colored populations, and for various tri-racial hybrids. Similarly, extensive incorporation of Amerindian genes into the American Colored population can be ruled out by serological data. With such information on hand, deviations from expected proportions of various gene-determined traits, such as

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* Both in his earlier book, and in his Boston University Lecture, Boyd's system of "races" properly involved serological descriptions of natural populations rather than serological races based on blood-group frequencies alone.

the abnormal haemoglobins, may then be attributed to natural selection.

But the blood groups themselves are subject to selection. Unquestionably, there has been selection against the Rhesus-negative gene, and against both A and B in favor of O, at least in Europeans. For these reasons, present gene frequencies do not provide a perfect indication of what they were in the past, and guessing as to proportions of groups entering into ancient admixture becomes a most hazardous activity. Valuable as they are in the study of contemporary populations, the blood groups are of limited value in solving ancient ties between races, but so are the conventional morphological traits.

SUMMARY

The serological factors (blood groups) discovered since 1900, have added tremendously to the scientific study of race. Simply inherited and susceptible to analyses as gene frequencies, the ABO, MNS, Rh, Kidd, Duffy, Diego, Kell and Lutheran systems of alleomorphs make possible detailed analyses and comparisons of contiguous local races, and help in reconstructing origins of recently-mixed populations.

As with classifications based on morphological traits rather than on the populations themselves, artificial "serological races" add nothing to human taxonomy. The major use of blood groups in classification is in the comparison and analysis of natural populations, and in the study of natural selection in contemporary races.

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* Reprinted in *Readings on Race.*

V

NATURAL SELECTION AND RACE

I_N THE *Origin of Species*, an epochal book published one hundred years ago, Charles Darwin described the principal mechanism of evolutionary change. Pointing out the inherent variability of all living species, and their tendency to depart infinitely from a central type, Darwin saw in natural selection the directive force behind each species, and smaller taxonomic units as well.

Natural selection provides a mechanism for change within species and especially within races. With natural selection in operation each race undergoes continual change. Ultimately, the descendants scarcely resemble their ancestors, phenotypically and genotypically as well. Though the many races of mankind derive from a common source, they have come to differ widely, both among themselves, and from their first *sapiens* progenitor.

Natural selection involves no mystery, no mirrors, no Lamarckian "will to change" and no Bergsonian *elan vital*. All of the climatic forces we see about us are potential selective agents bringing about, through genetic adaptation, differentiation between races. Food is a selective agent by its very abundance favoring sheer fertility, or by scarcity, favoring smaller size and slower growth. So is disease a powerful selective agent, favoring in each generation those with superior immunity. The natural world is full of forces that shape races and make one race different from another.

Thus, when we view a race, long-established in a particular climatic zone, habituated to specific foods and levels of caloric intake, and subjected to local diseases, our first inclination is to explore the immediate environment for selective factors responsible for local differentiation. Or, having caught sight of some

unusual difference, we may choose to investigate it alone and to
determine what adaptive advantage its presence or absence may
convey.

However, since local environments are multitudinous (and for
the most part ill-explored), it becomes quite a task to assess the
race-making potentiality of a bog here and a swamp there. In-
dividual human differences, numbering in the thousands, present
a comparably formidable task to analyze. How do we measure the
evolutionary significance of the inner or "Mongolian" eyefold,
the reasons for a given gene frequency among the Tlingit, or the
reasons for peculiar Duffy and Diego frequencies among the
Lacandons?

Here the broad-gauge view comes into its own. Instead of
trying to match each racial difference to its corresponding selec-
tive advantage, we often search for broad generalizations involv-
ing variables of climate or major somatic differences. We look for
characteristics particularly associated with extreme heat or cold.
We look for climatic variables differentially associated with body
build or skin pigmentation. We watch for diseases that may
explain the distribution of the abnormal haemoglobins. In short,
and detective-wise, we seek for clues that may explain particular
directions of racial differentiation, as well as the process of race-
making in general.

ENVIRONMENTAL DIFFERENCES

One major environmental difference is temperature, and with
it the intensity of solar radiation. World extremes run from 130°F.
in the shade, to minus 60 and below, a range of 190° F. Usually,
the extremes are found in very different regions, but in the desert
soil-temperatures may drop from 125°F. at midday, to below
freezing the very same night. The same man may come near to
blistering his feet at noon, and frosting his toes in sleep. This is
true in the Kalahari, in the deserts of Australia and in the deserts
of the American southwest.

Solar radiation, contributing to the radiant heat load varies
by a factor of nearly 10,000. An exposure meter may go off-scale
in the desert and barely yield a reading in the Arctic night-day.
It is true that houses, umbrellas, central heating and air-condi-

tioned rooms mitigate the extremes for many of us now, but this
is not true for all, and such protection against nature is relatively
recent in man's million-year existence. When we read of death by
sunstroke or by cold, we may well realize how much more strin-
gent climatic selection was a hundred and a thousand years ago,
and what a large proportion of the population faced climatic ex-
tremes with relatively little environmenal protection.

We in America and Western Europe have unlimited access to
food. With no additional cost we could become circus fat-men,
ingesting a daily 6,000 calories instead of our national averages
of 3,000 to 3,500 calories a day. Overnutrition is clearly a major
cause of death with us: as a population we are adapting to a food
surfeit. But in many parts of the world, 1,500 calories a day (a
weight-reducing value for us), is a feast, and 800 calories per day
is unfortunately "normal" for many. Our breakfast of bacon and
eggs and buttered toast, and coffee with cream provides as much
meat as many people get in a week, and is comparable in fat
to several days' food for them. An American adult drinks more
milk in a few days than most African and Asiatic toddlers see in
a year. As with temperature, extremes in food intake are great.
Death by starvation is in fact far more common than death by
heat or cold.

Besides temperature and food, there are numerous infectious
diseases that slaughter people by the billions. In Lincoln's time,
Americans died in great numbers of cholera, typhus, malaria,
diphtheria, scarlet fever, pneumonia, tuberculosis and dysentery.
Quite recently, Eskimos succumbed to measles and mumps and
whooping cough. In other parts of the world malaria, elephanti-
asis and yaws, amoebic dysentery and snail carried Bilharzia are
common causes of death. Little wonder that in some primitive
areas today scarcely one infant in three lives to adulthood. And in
each part of the world, a unique combination of pandemic diseases
contributes mightily to race differences.

PIGMENTATION AND NATURAL SELECTION

Variations in skin pigmentation in man have obvious adaptive
value. As most of us know from personal experience, even a little
melanin in the outer layers of the skin is protective. Early in the

season, before we have begun to tan, even short exposures to bright sunlight may result in discomfort. Later, with a good tan, we can gambol for hours in the August sunshine.

Darker skins represent natural protection against the most damaging portion of the solar spectrum, the ultra-violet and especially about 2200-2800 Angstrom units. Not only is more melanin protective against immediate damage to the deeper layers, but it has a long-term value as well. Skin cancer is more common among "whites" in Texas, than in more northern parts of the country, and there are conspicuous Negro-white differences in the incidence of skin cancer, even with correction for occupation.

Balanced against the advantages of pigmentation in brighter climates, are possible disadvantages in duller climes. Vitamin D, essential to bone growth, is produced by the irradiation of ergosterol in the skin. Heavy skin pigmentation could be disadvantageous where the D vitamin intake is low leading to childhood rickets and birth complications.

Protection, on the one hand, and interference with vitamin-D synthesis on the other may well explain the north-south gradient in skin pigmentation from Finland and Denmark to the Equator, and the smaller but equally apparent gradient in Asia. In addition, one must note the contribution of skin pigmentation to the amount of radiant energy converted into heat in the most superficial layers of the skin. With darker skin reflecting far less of the visible and near-UV portion of the solar spectrum, American Negro skin temperatures are higher, under standard conditions of comparison and the inner (rectal) temperatures are distinctly higher as shown by Paul T. Baker (1958). This would raise the heat load to be dissipated, but could be advantageous where water supplies were adequate. In fact, the relatively lighter skins of the Bushmen and Hottentot may be viewed as a compromise.

BODY SIZE AND NATURAL SELECTION

Among the many differences between races, variations in body size are especially conspicuous. In some groups males average close to six feet, and in other populations average male stature is nearer to five feet. The *fat-free* body weight of American males approximates 135 pounds (some go as high as 190 pounds), while

the comparable *fat-free* weight in other groups may average as little as 105 pounds.

Large body size can be advantageous. It commands respect, it is helpful in wrestling and hand-to-hand fighting, and it is a useful adjunct in hunting big game. The bigger man can cover more territory, he is speedier, he can tackle bigger game and bring back larger cuts of meat. Not too surprisingly, the noted hunting peoples of North America and Europe have been tall on a world scale. Given large animals to hunt, size is adaptive.

But size and massivity have their disadvantages. Larger size requires more calories, merely to keep alive, as Americans, Dutchmen and Englishmen learned in Japanese concentration camps. Larger size requires more calories to grow on so that the genetically-large child is at a particular disadvantage when food is scarce. And the large man, while more efficient at heat regulation in cold weather, is less efficient in hot weather.

For small size, the advantages and disadvantages reverse. Size is of no advantage when tending a trap. The less food there is, the more advantages accrue from being sub-sized. On short rations the genetically-small child has a better chance to live and come to maturity. In the extremes of heat, the small man is unquestionably favored, as is true also during violent exercise even at moderate temperatures.

The small peoples of the world tend to be found nearer the equator, and there is a marked *negative* correlation between the mean annual temperature and weight. As one moves southward in Europe temperature rises and weight drops, as D. F. Roberts (1953) has demonstrated for 116 different populations the world around. The very lowest average weights (96-100 pounds) are associated with mean annual temperatures of 70-82° F., the highest average weights (in excess of 160 pounds) are associated with a mean average temperature of 40° F. (Fig. 12).

As with any statistical association, the relevant variables are undoubtedly complex. Part is unquestionably physiological adaptation. Russell Newman (1956) has demonstrated for America that the colder the state of origin, the more fat young men have! Part is in all probability genetic adaptation to extremes of heat and cold with the little men stemming from lands that are hotter.
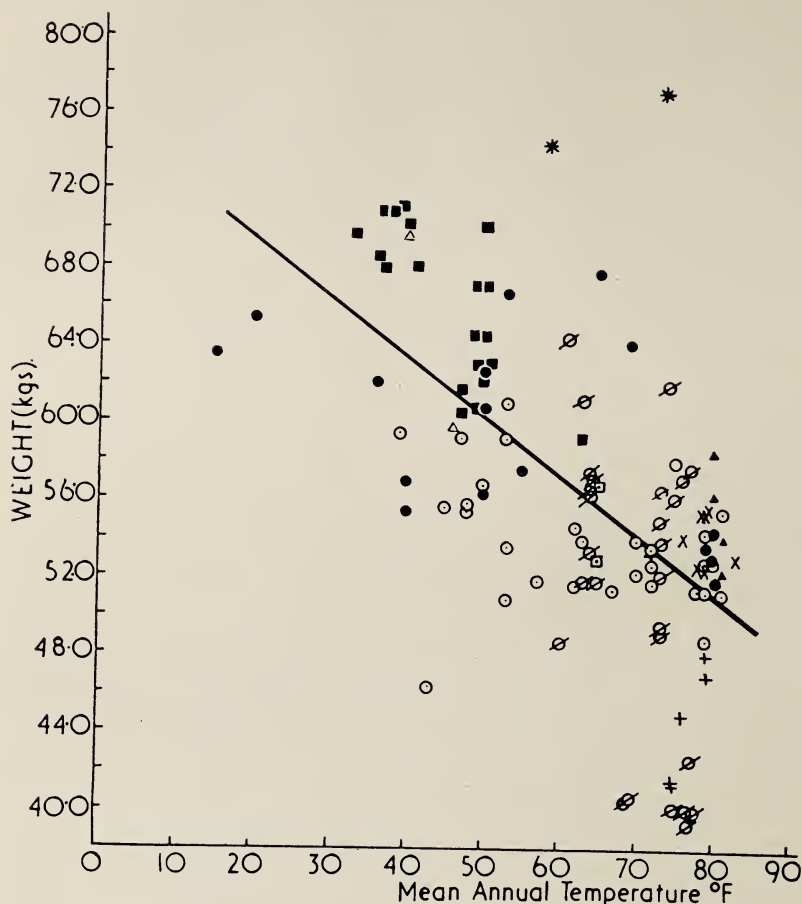
Fig. 12. Relationship between mean body weight and mean annual temperature. The higher the mean annual temperature, the lower the body weight of populations tends to be. (From Roberts, 1953, and Garn, 1960.)

And part may be attributed to genetic adaptations to nutritional extremes in the overcrowded, "underdeveloped" equatorial lands. Famine is a powerful selective force, differentially eliminating the massive and large. Famine does not have to be a consistent part of the environment; one dies but once of starvation. In the face of continual caloric restriction, the genetically-small individuals have a better chance to reach maturity and to reproduce themselves.

Within each geographical race there is a range of sizes small to large. This range can best be comprehended in terms of the selective forces, temperature for one, and the available food supply for another. However, these generalizations about body size are fully documentable but do not explain the Ithuri Forest Pygmies. Nor do they explain the surprising fatness of the Papago Indians of North America. These particular exceptions merit very particular investigation and the metabolism of the Pygmies is currently being investigated by Dr. George Mann.

BODY-BUILD AND NATURAL SELECTION

Design a man for extreme cold and you have a virtual globule. Thick-set, reduced of leg and peripheres, there is a minimum of outstanding projections. By limiting surface relative to mass, less heat is loss by radiation, conduction and convection and (probably) by insensible perspiration. By pulling-in projections closer to the warm body core, differential cooling is avoided with consequent danger of freezing.

Design a man for dry-desert heat and he approaches paper-thinness. By maximizing surface relative to mass, heat loss by perspiration is maximized. Long hands and feet and a beaky bony nose, while contributing relatively little to the total cooling surface, are at no disadvantage where environmental temperatures are high relative to body heat.

It is not quite appropriate to bring in a variety of species for comparison, selecting perhaps the rotund seagoing mammals at the one extreme, and the linear desert fox at the other. Nevertheless, desert-adapted representatives of a species do tend toward linearity while related arctic forms exhibit a lower surface-mass ratio (Bergmann's law). And, in man, there is a distinct tendency for the more linear groupings to be associated with high (and relatively dry) environmental temperatures, and for the thickest populations to inhabit areas where below-freezing temperatures predominate.

Adaptations in body-build, however, involve more than just proportions, complicating the analysis of what we readily see and most easily measure. We must consider body composition, the amount of fat and especially the thickness of the insulating

Fig. 13A

Fig. 13A & B. Extreme differences in body proportions and the surface/mass ratio in Eskimos and in the Nuer. (Photographs, courtesy of the National Museet, Copenhagen and Evans-Pritchard, E.E.: *The Nuer,* Oxford, The Clarenden Press, 1940.)

Fig. 13B

blanket of outer fat. We must consider the location or "patterning" of the outer fat, which is subject to sexual as well as climatic selection. Fat over the cheekbones of Eskimos, even thin ones, and about their orbits, not only contributes to their flat-faced appearance, but constitutes a protective mask as well. The stored fat on the rumps of Bushmen and Hottentots, an exaggeration of the typical hominid pattern, constitutes a particularly neat solution to two different problems. It provides a caloric reserve, an energy store for use during periods of food-scarcity, and being restricted in its location, least interferes with heat loss to the environment.

Variations in body proportions, in the amount and patterning of fat, and in other bodily constituents (including the plasma volume and red-cell-volume) are not the only cold-climate and hot-weather adaptations. As we have come to learn, each environmental stress is met by multiple adaptations. But racial variations in body build are conspicuous and we are beginning to learn much about the "inner contours" of people of various races. Moreover, experimental studies can be devised to test their adaptive values, to verify the directions that natural selection has taken in shaping the shapes of man.

ADAPTATIONS TO EXTREME COLD

Of the climatic extremes into which man has ventured, none is more quickly lethal (and therefore more selective) than the extreme cold. Winter temperatures from minus 40° Fahrenheit to minus 90° F. are reported for various places in the inhabited Arctic. Without well-designed winter gear, such temperatures would be deadly in a matter of minutes. Even in full arctic regalia frostbite and death by freezing are ever present potentials, their severity unalloyed by the fact that temperatures within the snow-built igloo or sod barbara may be high.

One obvious adaptation to cold-weather living is a minimum surface/mass ratio, with its obvious heat-conserving potentiality. A second involves size reduction in the limbs, particularly the legs, bringing them closer to the warm, central core. Both are characteristic of arctic peoples (Fig. 13). An increased thickness of subcutaneous fat would also be energy-conserving as Baker and Daniels (1955) have confirmed. However, Newman (1956),

working with draftees, has indicated that such a response may be largely physiological rather than purely genetic. Of the various cold adaptations known in mammals, the ability to generate more heat by raising the metabolic rate would be applicable to man, provided of course, that enough food is available. Evidence from a number of experiments favors enhanced metabolic responses to cold in Eskimos (Brown and Page, 1952), and in certain other groups.

Despite warm arctic clothing, the face is still exposed to wind and cold. The hands must be protected against freezing and must maintain their finer manipulative skills. Moreover, the peculiar Eskimo physiognomy (much more consistent across the Arctic than Eskimo serology) must also be noted in this connection. With fat-padded malars, fat-filled orbits, narrow eye-slits and reduced nasal profiles, the Eskimo face fits the model of a cold-weather face. And a number of studies, like those of Brown and Page (1953) confirm higher peripheral skin temperatures during cold stress (Barnicot, 1959). By implication at least, manual dexterity in Eskimos would be less impaired at low environmental temperatures.

It should be emphasized that studies on cold adaptation have been so far limited by technical difficulties, subject selection and sample size. Laboratory experiments, moreover, fail to simulate natural selection where sheer survivorship rather than peripheral skin temperatures is the test of adaptive fitness. Nevertheless, the probability is that peoples long exposed to cold-selection will exhibit, on the average, features and mechanisms of value in the cold.

ADAPTATIONS TO NIGHT COLD

In contrast to the Arctic and its far below-zero temperatures, minimal night temperatures at near-freezing levels (or slightly below) may seem unimportant. But such frosty situations command a very large part of the world, even the simmering deserts. And there are many human groups (quite recently some of our ancestors) who, poorly-clad, were in danger of frosting their toes as they slept. The problem of peripheral skin temperatures at moderate levels of coldness further relates to American Negroes,

who apparently experienced more cold injuries during the Korean conflict than other soldiers.

Among the groups studied to date are the Alacaluf (the aborigines of Patagonia), desert-dwelling Australian aborigines and Bushmen of the Kalahari. All exhibited the ability to sleep nude or partly nude, often using their clothing as pillows under conditions quite uncomfortable for Europeans. Generally, they exhibited an enhanced ability to raise their metabolic levels and thus generate more heat except for the aboriginal Australians. Peripheral skin temperatures tended to be higher than in European subjects under the same conditions (indicating greater peripheral blood flow). Again, the Australians' adaptations were in the reverse direction (see Fig. 14).

ADAPTATIONS TO HUMID HEAT

A hot-humid environment is especially enervating, as we know in the sticky dog-days of summer. With moisture-saturated air, even below 100° F., the body becomes bathed in a continuous layer of sweat in an effort to reduce the heat load. In the tropics, there is attendant danger of excessive salt loss. If the body temperature rises too high, there is the possibility of death by circulatory collapse. Even apart from this extreme direction of selection, hot-humid environments would tend to favor individuals who can maintain a moderate work load in the water-saturated atmosphere.

For the existence of heat adaptations in man there is both direct and indirect evidence. The poorer cold-adaptation exhibited by American Colored subjects in a number of studies would suggest that they are relatively heat-adapted instead. Paul T. Baker (1958) compared colored and white subjects, carefully matched and acclimatized, and concluded that the former were better adaped to *humid* heat (but not to dry heat).

Assuming that the long-term inhabitants of steamy climes are the product of selection by humid heat, what mechanisms are at least theoretically involved? Dark skin color, by raising the surface temperature, could bring about sweating earlier. An increased sweating rate involving more sweat glands has been suggested, but is currently the subject of debate as summarized by Barnicot (1959). A lower rate of salt loss is among the other
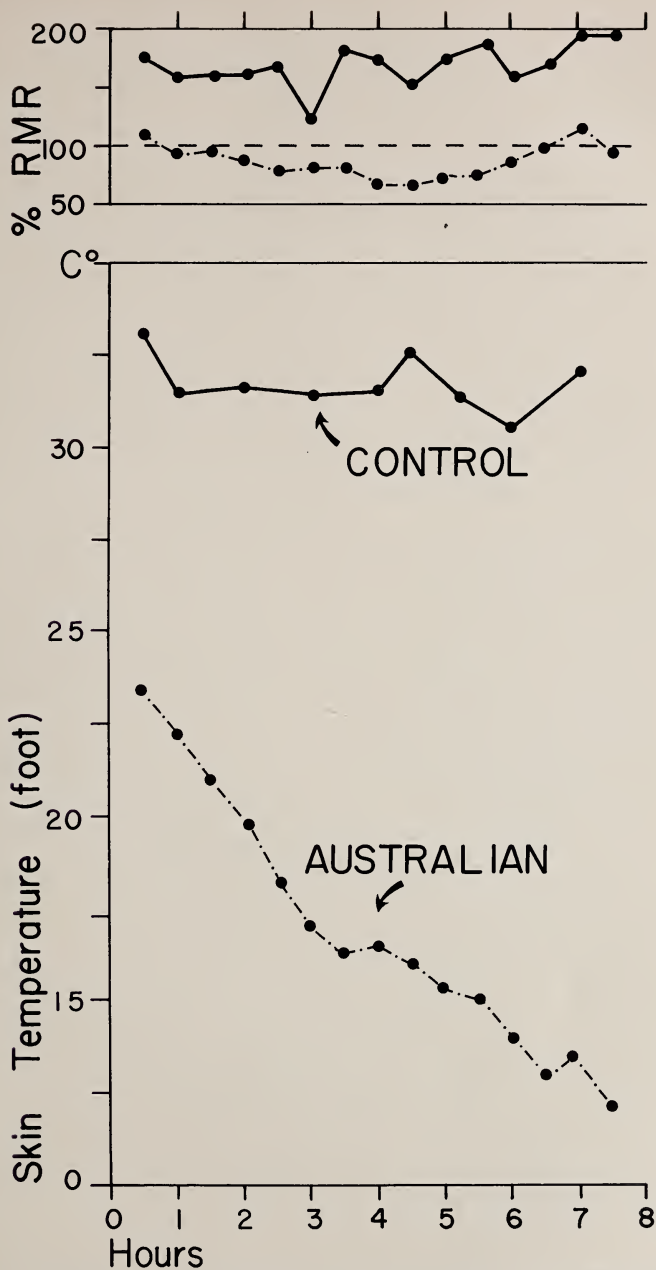
Fig. 14. Adaptation to moderate cold during sleep. In desert-dwelling Australian aborigines adaptation to near freezing night temperatures involves a decrease in peripheral skin temperature. The marked elevation of the relative metabolic rate shown in control whites is not exhibited by the aborigines. (From Hammel *et al.*, 1959.)

mechanisms currently being studied. A fourth possibility—known for some desert animals—is the ability to tolerate increased heat loads.

Logically, the peoples of the African rain-forest and those of the Amazon basin exhibit adaptations to humid heat, though not necessarily the *same adaptations*. For both groups, of particular theoretical interest, the direct evidence is at present minimal.

ADAPTATIONS TO DESERT LIVING

Desert living is essentially a compromise, requiring tolerance of mid-day heat and night cold, tolerance of high ultraviolet intensities without increasing the heat load, and the ability to lose heat by perspiration while conserving precious water.

Desert mammals suggest several pathways to arid living, including nocturnal habits, leanness and small body size. Only the latter two appear to be applicable to man, who notably ventures into the noonday sun. The early loss of subcutaneous fat in the Bushman may indicate its inadaptive nature under Kalahari conditions and the Bushmen are notably small, as are many other desert peoples.

In theory, skin pigmentation should be moderate in desert men, enough to protect the malpighian layer, but not enough to build up the heat load. Baker (1958) has shown the disadvantages of extremely dark skin under hot-dry conditions. Thus the lighter pigmentation of the Bushmen may represent the theoretical compromise situation, a balance between competing directions of selection.

A third area of desert specialization particularly exhibited by the Kangaroo rat *Dipodomys* is the increased ability to concentrate urine. If man could follow the lead of this desert-living rat, he could conserve one to two pints of water a day, not a great deal compared to water lost in the exhaled air, but a saving nonetheless. No evidence exists at present for or against the possibility of increased urine concentration in desert man, a possibility further complicated by their largely protein dietary. However, there are two disorders in which urine is *less* concentrated than usual: the first is fibrocystic disease of the pancreas. Heterozygotes for this disease, therefore, would be less viable under desert

SOME RACIAL DIFFERENCES IN RESPONSES TO HEAT AND COLD

Author	Sample	Findings
Hammel et al. (1959)	8 Central Australian aborigines 6 Control "whites" 9 Tropical Australian aborigines	Small but important differences in thermal and metabolic responses to moderate cold during sleep.
Scholander et al. (1958)	5 Australian whites 6 Central Australian aborigines	Europeans maintained body heat by increased muscular movement during sleep.
Wyndham and Morrison (1958)	2 Unacculturated Bushmen 2 Europeans	No important differences found in response to moderate night cold.
Hammel (1960)	9 Alacaluf	Markedly increase metabolic response to night cold, persistent elevated BMR.
Adams and Covino (1958) (see also Brown and Page (1952)	7 Negro soldiers 7 "Caucasian" soldiers 6 Anaktuuk Eskimos	Systematic Negro-white Eskimo differences in skin temperature and BMR during 2-hour exposure to cold.
Lampietro et al. (1959)	17 white and 16 Negro volunteers matched for body size and composition	Fewer rewarming cycles and lower finger temperatures in Negro subjects exposed to moderate cold.
Bass et al. (1959)	16 East Indians 16 U. S. Negroes 23 Chinese 17 U. S. Whites 8 Eskimos	Marked racial differences in plasma volume and blood volume especially for Eskimo of questionable relationship to climatic adaptation.
Baker (1958)	40 pairs of white and Negro soldiers matched for body composition and size	Negroes displayed a higher physiological tolerance to hot humid conditions, but heated up more under hot dry conditions.
Wyndham et al. (1952)	8 African mine laborers White data from the literature	Lower sweating rates, lower heart rates and lower rectal temperatures for Africans
See also Barnicot (1959)		

conditions, and it is a safe bet that the gene involved will prove rarer in the hotter, dryer parts of the world.

The second gene-determined condition known to be unfavorable in the desert is the sickling trait (see chapter VI). Peculiarly enough, the heterozygote *NS* has a reduced ability to concentrate urine, and is therefore at an adaptive disadvantage where water is scarce (Keitel, *et al.*, 1956). Needless to say, the sickling trait uncommon in the less watered parts of Africa and Saudi Arabia, in part because the heterozygote is at no advantage in such areas, and in part because the trait is disadvantageous where man's water loss is especially critical.

SUMMARY

Increasingly, population differences in body size, body-build and proportions and skin pigmentation may be viewed as adaptations to particular climatic extremes. Such differences often transcend geographical races while the distribution of the differences provide a hint as to the origin of particular races.

However, a particular climatic stress will not always be met by the same adaptation. Therefore, the adaptive nature of racial differences must be determined under conditions simulating the environments in which they have arisen, and using acclimatized controls.

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VI

ABNORMAL HEMOGLOBINS, MALARIA AND RACE

For generations mankind has lived with malaria, or to be more exact, has lived despite malaria. Great sections of the world have proved unhealthy, malarious, poor places to survive In both hemispheres, countries situated between 35° N and 20° S have been cradles of malaria, the "shaking ague." In Europe and the Middle East malaria had control of both sides of the Adriatic, much of the Italian coastline, Greece, the Ionian Islands and Crete, and the shores of the Black Sea and the Caspian Sea. Since the malarial parts of the world, including the New World, were generally well-watered and usually densely-populated areas, it is not surprising that in 1880 one half of the entire mortality of the human race was attributed to malaria!

In the areas of endemic malaria, in southern Europe and East Africa, it was observed that some few individuals did not acquire malaria, or at least did not exhibit the chills and recurrent high fevers, the enlargement of the spleen and intestinal symptoms characteristic of the disease. Various explanations were offered, especially in the days when malaria was blamed on night air and exhalations of the marshes. Still, no really satisfactory evidence was offered as to whether immunity to malaria was natural or acquired early, whether it was due to some chance event (such as being unattractive to mosquitoes) or whether malarial immunity existed at all.

Rather recently, and as a by-product of studies on hereditary blood disorders, the existence of true natural immunity to malaria has been confirmed. Even more important, the relevant mechanisms have been discovered, and the disease of malaria has been shown to be responsible for several important directions of polymorphism in man.

THALASSEMIA AND THE MEDITERRANEAN

The story begins in the Mediterranean, in Italy and Sicily, Greece, and some parts of the Middle East. There, the disease called *thalassemia* is common, a disease that has two distinct forms, *thalassemia major* and *thalassemia minor*.

Certain interesting features of thalassemia soon made themselves evident. The first, of course, was the Mediterranean distribution of the disease, especially (as noted in the United States) among individuals of Italian or Greek ancestry, but also among North Africans, Egyptians and peoples from Asia Minor. Though observed elsewhere in the world (for example, Thailand) but rarely among Europeans not from the Mediterranean area, thalassemia is for practical purposes primarily limited to the Mediterranean and Irano-Mediterranean local races of the European Geographical Race (see chapter XI), and to local races in India.

THE MECHANSM OF THALASSEMIA*

Clinical status	Genotype	Clinical picture	Hemoglobins
Normal	Homozygote normal	Within normal limits for hemoglobin and cellular fragility	Normal, slight amount of fetal hemoglobin
Thalassemia minor	Heterozygote	Slight anemia, increased osmotic pressure of the red cells	slight amount of fetal hemoglobin
Thalassemia major	Homozygote	Marked anemia, abnormal red cells fragile and increased osmotic pressure	Hemoglobin primarily of fetal type, little normal hemoglobin

* From Ingram and Stretton (1959), Neel (1949, 1950).

Family-line studies show that thalassemia is hereditary and that parents of a person with thalassemia major are "carriers" of the disease and exhibit thalassemia minor. Thalassemia is inherited as a dominant with thalassemia minor the heterozygotic state and thalassemia major the homozygotic. From the observed incidence of the disorder it is possible to calculate the gene frequency which ranges from nearly zero in Switzerland and northward in Europe

to 0.20 or more in certain areas of Cyprus and northern Italy. For much of Italy, where epidemiological data is quite complete, frequencies for thalassemia average under 0.02, from 0.02 to 0.06 in Sicily and Corsica, 0.10 in the coastal area northeast of Bologna and 0.18 in the Ferrara region.

THE GENETICS OF THALASSEMIA

Epidemiological and family-line data together confirm the existence of thalassemia as simply inherited in the form of a Mendelian dominant and with gene frequencies exceeding 0.02 and sporadically as high as 0.20 in the Mediterranean coastal area. Biochemical studies on the blood of individuals suffering from thalassemia major (the homozygotes) and other studies on the heterozygotic individuals suggest that the defect is primarily in the ability to produce normal adult hemoglobin A. Thus, the blood of sufferers from severe Cooley's anemia is almost entirely of the fetal type (or possibly of the A_2 hemoglobin subtype) while in thalassemia minor, the proportion of fetal hemoglobin is low. In short, the homozygotes cannot produce adequate amounts of the normal adult hemoglobin A, and their symptoms are largely referable to this gene-determined hemoglobin deficiency.

It must be noted that the individuals homozygous for the abnormal hemoglobin, that is sufferers from thalassemia major, rarely reach reproductive age. In fact, Neel (1950) doubts whether the homozygotes could reproduce even if they lived to the child-bearing period. This lethality obviously eliminates some proportion of the thalassemia genes in every generation.

THALASSEMIA AND MALARIA

The clinical and geographical data on thalassemia raised two important problems, one being the origin of the abnormal gene (and its seeming restriction to the Mediterranean) and the other being its continuance in time. From the wide distribution of the gene on both shores of the Mediterranean, an ancient origin could be postulated—prior to the spread of the Mediterranean local race as early as 5000 B.C. However, the absence of thalassemia in parts of Europe where Mediterraneans have migrated remained a puzzling feature. Even more peculiar was the continuation of

the abnormal gene. Since, in each generation, a certain proportion of the abnormal genes are lost, the mutation would ordinarily be expected to remain at a low level—balanced only by new mutant genes.

What then, explains the continuation of the abnormal gene, and its exceptionally high incidence in Crete, Bologna and Corsica? One explanation would be an increased fertility in heterozygotes, but such increased fertility has not been found and could not explain known "islands" of thalassemia. Similarly, an abnormally high mutation rate for the thalassemic gene, though postulated, has not been found, and again would not explain the marked variations in the incidence of thalassemia in Italy, Greece and elsewhere from Iran to South China.

Beginning in 1950, however, a number of workers pointed to the correspondence between the distribution of thalassemia and the incidence of malaria. In those parts of Europe where malaria was holoendemic, that is a severe problem the year round, thalassemia frequencies were highest. In the cold-climate and higher areas, thalassemia frequencies drop to near zero. Working in Sardinia, Cepellini (1955) has shown a far higher incidence of thalassemia in low-lying regions where malaria is holoendemic than in corresponding villages at higher altitudes. The emerging picture, and one now well supported by findings on the sickle-cell disease which follows, is that the thalassemic gene affords protection against malaria.

In a malarial region the homozygotes for thalassemia die early without reproducing. The "normal'" homozygotes are afflicted with malaria and frequently die early, while the heterozygotes are somewhat protected and thus the continuance of both genes is assured. This simple explanation fits the data, explains the non-uniform distribution of thalassemia in the Mediterranean and makes the race-limited nature of thalassemia comprehensible in terms of natural selection in malarial areas.

SICKLE-CELL DISEASE

A second hereditary blood disorder, primarily African in its distribution, is called sickle-cell anemia, after the characteristic appearance of the red blood cells in saline solution. As with

thalassemia, sickling is inherited as a Mendelian dominant and there are two forms—the mild sickle-cell *trait* (the heterozygotic state) and the severe sickle-cell *disease* (the homozygous state).

The inheritance of sickling was confirmed by Neel in 1949 who demonstrated that individuals with sickle-cell *disease* were invariably the offspring of two parents with the sickle-cell *trait*. The molecular nature of sickling, in turn, was reported by Linus Pauling in the same year and the existence of a number of abnormal hemoglobins was subsequently discovered by Neel and his associates. Briefly, the individual homozygous for the sickle-cell trait produces the abnormal hemoglobin S (or some other abnormal hemoglobin) which has less oxygen-containing ability than the normal hemoglobin A. In a typical East African population, as in the American Colored population, there are three genotypes—the homozygous "normals," the heterozygous individuals with the sickle-cell trait and those homozygous for the abnormal hemoglobin, exhibiting the sickle-cell disease (Fig. 15.).

In Africa, frequencies of the sickling trait vary widely from zero in some areas to as high as 40% in other areas. Initially, these marked variations were explained in area-incidence terms (assuming the origin of the gene in the areas of highest concentrations) or by postulating migrations from high-sickling areas to low-sickling areas. Such explanations, reminiscent of racial anthropology of the last century, and early explanations given for variations in the blood-group frequencies, put maximum weight on hypothetical migrations and minimum weighting on purely local, epidemiological factors.

However, as with thalassemia, there had to be a more dynamic explanation for both the continuance and the peculiar distribution of the sickle-cell trait than migration theory offered. Since the sickle-cell disease is usually lethal, and since there is a differential elimination of individuals with the sickle-cell *trait* (cf. Raper, 1949), some selective advantage had to maintain the sickling gene. A number of workers, among them Allison (1954) noted the geographical association between malaria and sickling (Fig. 16.) and postulated an adaptive advantage for the heterozygote in malarial areas. This postulate has now been well documented in a number of ways as reviewed by Allison in 1955.
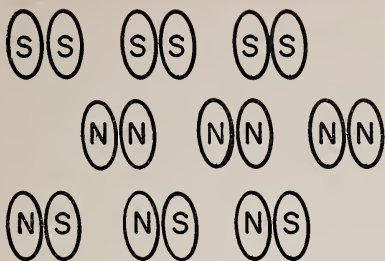
IN AN EAST AFRICAN
POPULATION THERE ARE
BOTH SICKLING AND
NORMAL GENES.

IN EACH GENERATION
"SICKLER" HOMOZYGOTES
DIE OF SICKLE CELL
ANEMIA — —

NORMAL HOMOZYGOTES
DIE OF MALARIA — —

SURVIVING ARE THE
HETEROZYGOTES — —
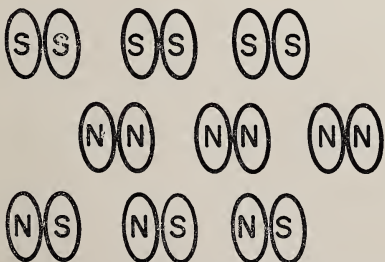
WHOSE PROGENY INCLUDE
SICKLERS, NORMALS, AND
HETEROZYGOTES, THUS
PRESERVING BOTH THE
NORMAL AND SICKLE GENES

Fig. 15. The mechanism of "sickling" and the differential survival of indi-
viduals with the sickling trait where malaria is a severe problem.

FALCIPARUM MALARIA: HYPERENDEMIC EPIDEMIC NO TRANSMISSION
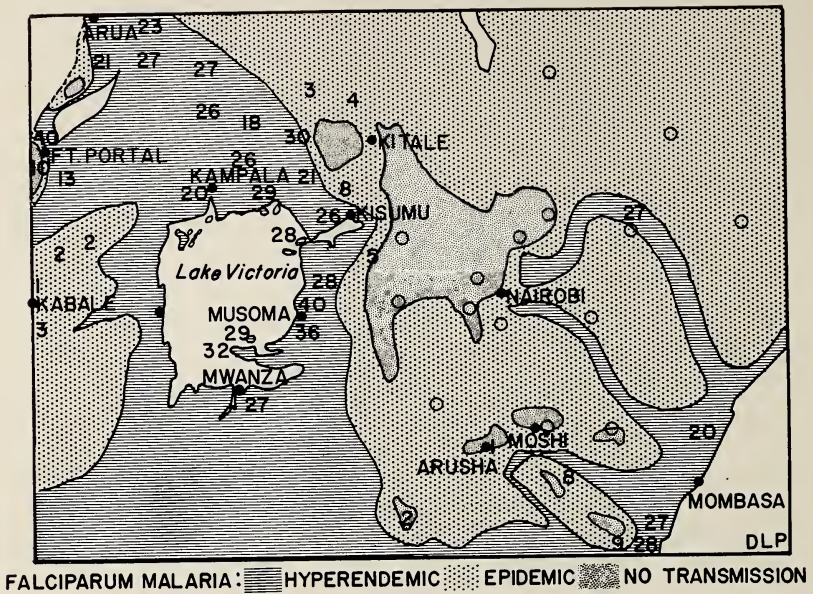
Fig. 16. Association between the frequency of the sickle-cell trait in East Africa (designated by the percentages drawn on the map) and the prevalence of malaria (as shown by stippling). Where malaria is hyperendemic the percentage of sicklers is highest. (Redrawn from Allison [1955] and reprinted from *Readings on Race*, p. 200.)

By now, the selection advantage attached to the sickling heterozygote has been confirmed, particularly by investigating children in African areas of hyperendemic malaria. In such areas, the homozygous *normals* develop malaria early, many die, and the vitality of the survivors is impaired. Those individuals homozygous for the sickle-cell trait develop sickle-cell disease with consequently increased mortality. The heterozygous sicklers, however, those with the sickle-cell trait but not the disease, are in effect protected. The incidence of malarial infestation is less. Heterozygotes develop malaria—but to a milder degree and with fewer effects particularly during pregnancy (Allison, 1955).

Thus, sickle-cell disease in Africa, like thalassemia in the Mediterranean (and in Greece where both are found) is an example of adaptive polymorphism in man. Both the normal gene, which serves to elaborate hemoglobin A, and the abnormal gene (which

is responsible for hemoglobins S, etc.) continue to exist in Africa because the heterozygote is at an adaptive advantage. The more common malaria is, the higher the gene frequency for sickling, reaching toward 0.50 as a maximum. But in areas where there is no malaria, or where malaria has come under control (as in western Europe and the United States) the heterozygote is at no advantage. Thus DDT, marsh drainage, screens, mosquito repellents and antimalarials will eventually reduce the incidence of the sickling gene.

It will be noted that the sickle-cell trait in Africa transcends the conventional boundaries of African local races. Even within related populations, the incidence of sickling may be far higher in one group than in another. To some extent, these local differences may be due to the presence of other abnormal hemoglobins (C, E, etc.). But the greater part of the diversity is more easily attributed to purely local factors, chief among them the prevalence of malaria. Confirmatory evidence comes from Saudi Arabia where the incidence of sickling is far higher among oasis-village dwellers than among the hill tribes from whence these same villagers stemmed. All of this adds to the mounting evidence that the genetic makeup of any population strongly reflects the conditions under which it lives.

CULTURE, MALARIA AND THE SICKLE-CELL TRAIT

While the relationship between malaria and hemoglobins is quite clear in Africa, the existence of malaria itself poses a certain problem. Much of Africa is not naturally malaria territory: in its pristine state Africa offered few places for the mosquito *Anopheles gambiae* to breed. Only where the rain forest has been opened up by primitive agricultural practices (or in modern rubber plantations) does *A. gambiae* have a chance to spread. In the rain forest, or on the shaded forest floor, the malaria-carrying mosquito does not breed, and in the untouched rain forest malaria does not exist.

Reviewing such information, Frank B. Livingstone (1958) has concluded that malaria is of recent introduction in West Africa, following the spread of slash-and-burn agriculture, the opening of the forest floor and the appearance of stagnant but not shaded

pools for the carrier mosquito to breed in. Man made Africa malarial by providing the kind of climate the *Anopheles gambiae* mosquito needs, and by raising the human population density to the point where there were always new individuals to infect and thus spread the chain of malaria. The dissemination of malaria in Africa would thus follow the spread of slash-and-burn agriculture and the abnormal hemoglobin-S would thus become adaptive in its wake. Sickling frequencies, particularly in West Africa, apparently bear this hypothesis out; peoples still pre-agricultural or late in attaining agriculture have the lowest incidence of sickling.

CULTURE, MALARIA AND THALASSEMIA

Livingstone has suggested that man paved the way for malaria in Africa; by cutting clearings in the jungle; man gave *A. gambiae* a place to breed and the resulting spread of malaria put an adaptive premium on the otherwise inadaptive hemoglobin-S. This would appear to be the first concrete example of the way culture, that is learned behavior, is capable of bringing about genetic change within populations.

The man-mosquito-malaria relationship in West Africa inevitably suggests a review of man and malaria in the Mediterranean. Seemingly, Sicily, Sardinia, the Po valley and Cyprus have no immediate parallel with Africa. There were no jungles to cut down and while there has been deforestation in some areas, swamps such as the Pontine marshes can hardly be considered as recent human artifacts except by silting-up of rivers.

Nevertheless, while man did not create the wet, humid marshy lands of the Mediterranean, he certainly did move into them for the practice of lowland agriculture. The early civilizations of the ancient world grew up along rivers with their seasonal floods and consequent stagnant pools. Oasis villages (where malaria is holoendemic even today) provided the basis for early stable populations. Primitive ditch-irrigation, wheel-and-bucket watering and crops specifically adapted to seasonal flooding supported the bulk of the populations. The consequent food surpluses make large population densities possible, and vastly increase the numbers of people subjected to malarial selection.

SUMMARY

In the Mediterranean area through to Iran, and in a broad area of Africa, there are two distinct hereditary blood disorders that are irrevocably linked to malaria.

Thalassemia (or Cooley's anemia) is a hereditary disease, a minor disadvantage in the heterozygote (thalassemia minor) but lethal in the homozygote (thalassemia major). The rather high gene frequency for thalassemia in the southern Mediterranean appears to be maintained by malaria, generation after generation.

The sickle-cell trait (involving the abnormal hemoglobin-S) similarly reflects adaptive polymorphism. In malarial areas of Africa the normal homozygote and the abnormal heterozygotes are both at a disadvantage; the heterozygote has peak adaptive fitness where malaria is holoendemic.

In all likelihood the spread of malaria in Africa, and therefore the abnormal hemoglobins, was brought about by man's opening the forest roof by slash-and-burn agriculture. In the Mediterranean, lowland agriculture may have maximized population exposure to malarial selection. In both regions changes in the genetical makeup of populations can clearly be attributed to particular cultural practices.

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VII

RACE AND DISEASE

Since races are natural units, reproductively isolated from each other and with separate evolutionary histories through time, it is not surprising that they differ from each other in a great many gene-determined respects. Considering the unique history behind each race, and the geographical and ecological uniqueness of its successive homelands, lack of differentiation would be remarkable indeed. Particularly in the random loss or chance acquisition of genes, each race represents a cumulative succession of accidents that could never be duplicated in millions of years.

These statements about racial differentiation, applicable to the "normal" genes commonly considered, pertain to abnormal or disease genes equally well. For the rare diseases, represented by but few carriers even in large populations, chance events could be prepotent. At a gene frequency of 0.01, 0.001 or even less, chance might easily eliminate a disease gene in one population while doubling or even tripling its frequency in a second. In one breeding population, the requisite disease mutation might never have happened, while in yet another population that mutation could have taken the form of a run.

Checking upon gene frequencies for rare diseases is at present no easy task. In most areas of the world, lacking hospital facilities and diagnostic skills, population comparisons are impossible. Even in the great medical centers, rare hereditary disorders may easily be missed, while other disorders may erroneously be considered race-limited or population-limited in their stead. Obviously, even elementary data on the comparative frequencies of the rare gene-determined disorders are to a large extent lacking today.

Nevertheless, there are numerous genetic disorders, either rare or lacking in most populations, that reach major proportion in a

few groups. The sickle-cell trait, mentioned in the previous chap-
ter with its counterpart sickle-cell anemia, is one example, as is
Mediterranean anemia (thalassemia major). For these gene-
determined diseases, with frequencies close to zero in most parts
of the world, trait frequencies reach 0.2 to 0.4 in a few restricted
areas. In both examples the abnormal gene is maintained because
the heterozygote is at an adaptive advantage.

A second class of disorders almost unknown in most parts of
the world, but uniquely common in one restricted area, is in-
creasingly coming to our attention. One of these unique diseases
is *Kuru*, an extraordinary hereditary neurological disorder appar-
ently restricted to Eastern New Guinea. A similar apparently
population-limited neuromuscular heredofamilial disease has been
reported from the Trust territory. In the Mediterranean area,
familial Mediterranean fever and *Favism* are not only population-
limited but promise to become included, along with the abnormal
hemoglobins, among the conditions clearly adaptive to the hetero-
zygotic state.

The third category, that of genetic diseases rare even in the
populations that have them is beginning to yield information.
Leprechaunism appears to be an "Irish" disease, in that all known
cases are of Irish origin or descent. *Tay-Sachs* disease and *familial
dysautonomia* are probably "Jewish" diseases. The most economi-
cal explanation, that of genetic drift, probably explains these
population-limited disorders. However, there are beginning sus-
picions that natural selection is also at work. By way of example,
differences between the Ashkenazic (European) Jews and the
Sephardic (Spanish-Portuguese and North African) Jews sug-
gest that local environmental factors are important even with
respect to the really rare hereditary diseases. One can easily
understand how a disorder, such as *congenital ectodermal dys-
plasia*, where sweat glands are largely lacking would be more
likely to survive in Stockholm than in Salerno.

Mutations being what they are, no disease can be considered
to be entirely race-limited. The same abnormal hemoglobin that
protects Italians in malarial areas also exists in Burma. The gene
for familial dysautonomia probably also exists in some Moslem,
Shinto and Buddhist groups but so far the disease has been re-

ported only among Jews. Nor should similarities in the occurrence of rare hereditary disorders necessarily indicate common ancestry. The distribution of the sickling gene in Africa, Madagascar, Yemen and Saudi Arabia we know now to be influenced by environmental selection. Thus, similarities between races in the same ecological zone, traditionally attributed to admixture, are often due to common directions of selection.

KURU: NATURAL SELECTION AND SORCERY

Our first example is *Kuru*, a remarkable disease apparently limited to the Eastern Highlands of New Guinea. Unknown to medical science until 1953, and quite unstudied until 1957, Kuru is well described by its pidgin-English name "skin-Guria" meaning shaking. Kuru is a progressive and incurable neurological disorder. Within a year after the onset of symptoms, the afflicted individual ordinarily dies.

Typically, the first sign of Kuru is incoordination. The victim begins to stumble, then increasingly he becomes less able to walk and involuntary tremors become more and more common. Soon he is no longer able to sit, and speech becomes unintelligible. Next the abilities to swallow food, to urinate and to defecate are no longer under the individual's control. Commonly, the full course of Kuru is run in a year or less, but the complete progress of this disease may take as little as three months.

Peculiarly, Kuru is limited to one group in Eastern New Guinea, the Fore, and some neighboring people among whom Fore women have married. It is known that far more women than men are afflicted by Kuru so that the female/male ratio among affected individuals is 14-1 or even higher through the third decade of life. Possibly 1% of all Fore natives are suffering from Kuru at any time, and in some hamlets as much as 50% of all deaths are due to Kuru.

In an effort to find a trace element, poison, or food deficiency responsible for the diseases, Fore food, Fore body paints and even Fore campfire smoke were tested. No trace element or rare earth that could serve as a nervous-system poison has been found. Moreover, Fore men living on Government dietaries outside of the Fore area also develop Kuru, so that Kuru is not a nutritional-

deficiency disease. All evidence points clearly to Fore ancestry, and not the environment in which the Fore live. On one side of the formidable Lamari river the Fore have Kuru, but on the other side the Kukukuku people who have not intermarried with the Fore do not have Kuru (Figure 17). To the south, the Yar do not suffer from Kuru, but Fore who have ventured through the forests into the isolated Yar territory do develop Kuru. Fore women who marry into the Keiagana, Kanite and Kimi tribes develop Kuru but their hosts do not, thus proving that the disease is not communicable.

Kuru appears to be an hereditary disease, inherited as a Mendelian dominant, but hormone-mediated. The gene frequency is estimated as approximately 0.37. In the female the homozygote develops *early* Kuru whereas the heterozygote develops late Kuru. Among the males the homozygotes die of early Kuru while the heterozygotes survive as do the homozygous normals. Since most of the heterozygous females live through the reproductive period and even those homozygous for Kuru (married early in life) manage to have children, the continuation of the abnormal Kuru gene is therefore assured.

Still, one may ask what keeps up the rather high gene frequency for Kuru. After all, in each generation, many Kuru genes are removed from the population. Even the progeny of individuals heterozygous for Kuru are relatively fewer. Since an abnormally high mutation rate for Kuru is a most unlikely possibility, some adaptive advantage for the heterozygotes may well be sought.

Carleton Gajdusek, the outstanding American authority on Kuru, observes that *"leprosy and yaws are less frequent here* (in the Fore) *than in many surrounding populaces who do not suffer from Kuru."* Obviously, if the Kuru-gene protects against either disease, it could counteract the loss of genes due to Kuru. Another possibility is that childhood mortality is lower in heterozygous individuals, although the statistics to date are not impressive. Still, a differential survival of heterozygous males in early childhood could help to balance the loss of Kuru genes in later life.

But the Fore have an interesting custom in regard to Kuru, a
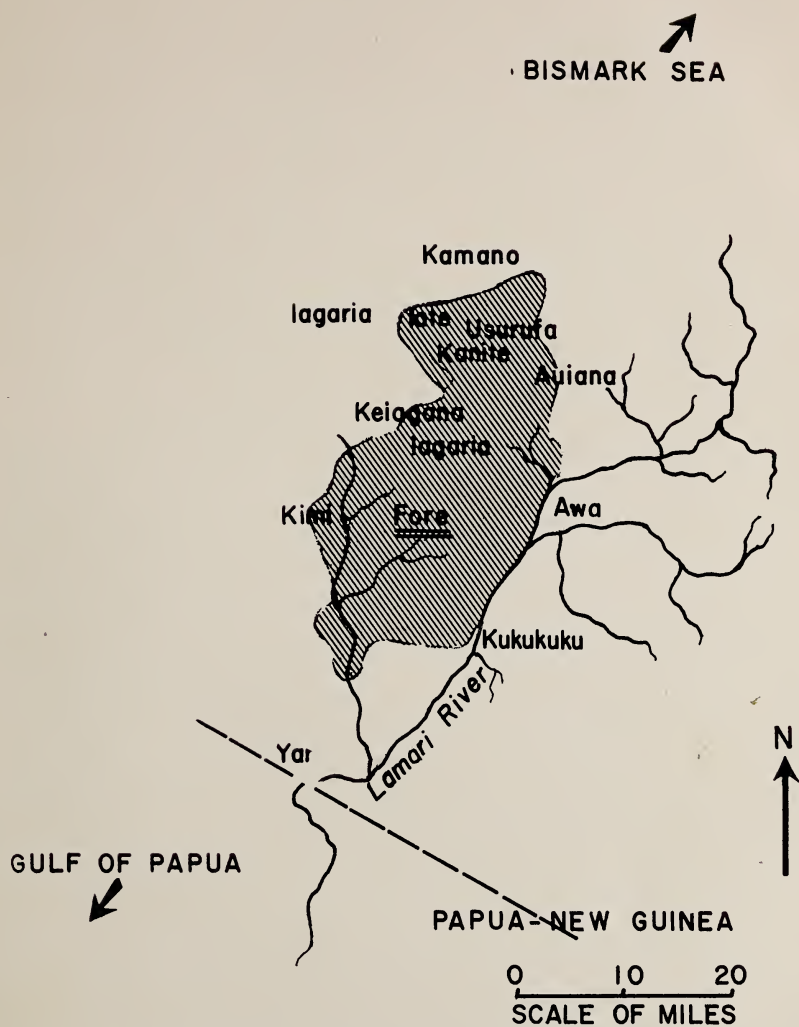
Fig. 17. The Fore territory in eastern New Guinea where Kuru is common. On the east side of the Lamari River Kuru is not found, whereas the gene frequency is as high as 0.38 among the Fore. (Redrawn from Gajdusek and Zigas, 1959).

custom that may explain the continuance of the gene. The Fore
think that Kuru is the result of sorcery. When someone dies of
Kuru, the Fore people seek out, attack and usually kill the sus-
pected sorcerer. This custom, called *tukabu* would have impor-
tant genetic effects. It would remove more normal genes than
Kuru genes since the ratio of normal to Kuru genes in the popula-
tion as a whole is 6:4. However, in adulthood the disparity is even
greater, with a continued removal of Kuru-prone homozygous
individuals. Thus, in adults, two or three normal genes might be
removed from the population for each abnormal gene lost to
Kuru. It may well be, then, that tukabu is a partial answer to the
continuance of Kuru among the Fore. Meanwhile the Territorial
Government has quarantined the entire Fore tribe.*

FAMILIAL MEDITERRANEAN FEVER

The borders of the blue Mediterranean overlook a long coast-
line, largely inhabited by a single local race. There, over great
antiquity, the great civilizations and the great religions of the
Western World arose. And in the Mediterranean there appeared
a rare hereditary disorder that deservedly bears the name, *familial
Mediterranean fever.*

Mediterranean fever is a "periodic" disease. Once the symp-
toms, the fever and malaise have begun, they recur sporadically
and unpredictably during the individual's lifetime. At the least
there is fever, lasting a day or two, joint pains and chest and
abdominal pain. In advanced cases, there is joint involvement,
decalcification of the bone and kidney insufficiency. Though most
of the affected individuals are not permanently or seriously dis-
abled, about 10% of cases studied to date succumbed to renal
complications.

The familial nature of Mediterranean fever is clear-cut. About
half of the siblings of index cases and about 50% of the offspring
of index cases develop Mediterranean fever, thus suggesting a
Mendelian dominant. There is a slight excess of males over fe-
males among the known victims but an autosomal gene is clearly
indicated.

So far a total of 249 cases of Mediterranean fever have been

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* *Science, 132*:77, 1960: *South Pacific Post,* May 24, 1960.

described in the medical literature. Of this total, at least 235 definitely stemmed from countries on or near the Mediterranean. The incidence appears to be particularly high among Armenians, Jews, Lebanese and Syrians as shown in the table below.

ORIGIN OF PATIENTS WITH FAMILIAL MEDITERRANEAN FEVER*

Stated Origin	Number of Patients
North America	67
Libya	25
Egypt	5
Israel	2
Syria	1
Lebanon	24
Iraq	22
Armenia	58
Cavasus	4
Turkey	14
Bulgaria	2
Greece	3
Italy	3
Spain	5
Not stated	18

* From Heller, Schar and Sherf ('58).

Interestingly, among 73 Jews with this disease studied in Tel Aviv, not a single one was Ashkenazic (the group including most European Jews). Heller, Schar and Sherf ('58) also observed that Sephardic (i.e., North African and Oriental Jews) contributed all of the Jewish cases described by other authors.

The facts are not incompatible with the idea that the mutation for familial Mediterranean fever is an ancient one in the Mediterranean as judged by its present wide dispersion. The mutation could well be 5000 to 6000 years old. The apparent concentration of the gene in the Eastern Mediterranean could be an indication of the site of origin, using the age-area approach, or it could be due to differential selection in the Bible lands.

Since the gene frequency of familial Mediterranean fever is low, well under 0.0001, the present limits may well be due to chance. However, since familial Mediterranean fever is definitely associated with impaired fertility, and a certain proportion of the genes are removed from the population in each generation, there

is need to explain how the gene frequency is maintained. The fact that Ashkenazic Jews do not exhibit the disease, while Sephardic Jews do, further requires explanation. A very likely possibility is that the heterozygote is at an adaptive advantage within the Mediterranean region, but not in northern, colder climes.

PRIMAQUINE DRUG SENSITIVITY

Further information on racial differences in physiology and disease came from testing synthetic antimalarials during and especially after World War II. Occasional individuals proved "sensitive" to the antimalarial drugs, that is they developed haemolitic anemia due to the destruction of red blood cells. The characteristic symptoms were demonstrable in occasional individuals who had been given primaquine (a quinine-like drug) such common drugs as acetanlid—often used in cold remedies—and the sulfanilamides. A small proportion of individuals maintained on doses of primaquine or acetanlid exhibited drug sensitivity but the vast majority of individuals did not.

When the proportion of drug-sensitive individuals was investigated in different geographical races, interesting results immediately appeared. The incidence of multiple-drug sensitivity in American "whites" proved to be low, 1% or even less. Among 1000 prisoners treated with primaquine and described by Dern, Beutler and Aving ('55) the incidence of sensitivity was 0.1%. However, a larger proportion of American Colored prisoners developed haemolitic anemia following the drug treatment. Among them, proportions of sensitive individuals ranged from 5% in one test to 11% in another group and 12% in yet another group. The true incidence of drug-sensitivity in the American Colored population is therefore close to 10% as shown in the following table.

DRUG SENSITIVITY IN WHITES AND NEGROES

Group	Drug	Per Cent of Sensitivity
491 "white" patients	Sulfanilamide	1.3%[1]
131 Negro patients	Sulfanilamide	12.0%
1000 "white" convicts	Primaquine	0.1%
199 Negro convicts	Primaquine	11.0%

[1] Calculated by Beutler et al ('57) based on data by Wood.

Studies on the blood of drug-sensitive and drug-insensitive individuals have shown marked differences in the stability of blood cell glutathione. To quote Beutler, Robson and Buttenwieser ('57) "When primaquine was administered to non-sensitive subjects there was no change in the red cell GSH (reduced glutathione) level. When primaquine was administered to a sensitive subject, however, there was an abrupt fall in the GSH content of the red blood cell to about one-half of the original already subnormal value." Thus the GSH content of *sensitive* red blood cells is less to begin with, and falls further upon administration of primaquine or some similar drug.

While the haemolitic anemia following drug administration to a sensitive subject is ordinarily of minor concern to the general practitioner, the far greater incidence of drug sensitivity in Negroes must be considered. The protection of Negro troops in malarial areas becomes a complicated matter, since approximately 10% of them are sensitive to the drug that protects them against malaria.

Moreover, the possible relationship of this primaquine type of drug sensitivity to endemic malaria becomes important to consider. If the drug-sensitive individual *already* has a reduced oxygen-carrying capacity in the blood, high parasitization for Falciparium malaria may not take place. As with the abnormal hemoglobins, his erythrocytes simply may not maintain the microorganisms at a critical phase in their reproduction. Thus the drug-sensitive person may be relatively immune to malaria, and the differential distribution of this trait in individuals of African ancestry may be a clear indication of both where and how the trait arose, as Motulsky (1960) has shown.

Primaquine-sensitivity is caused by a deficiency of the red-cell enzyme glucose-6-phosphate dehydrogenase. This type of deficiency has been shown to be particularly common where malaria exists. From Spain to the Philippines the incidence of this hereditary sex-linked blood-enzyme deficiency matches the prevalence of malaria. As with the abnormal hemoglobins, primaquine-sensitivity marks the impact of malaria on racial genetics.

FAVISM: WHEN GENE MEETS BEAN

Another disorder involving red-cell destruction is *Favism*.
Favism is an allergic-like response to the broad or Fava bean.
Susceptible individuals may develop hemolytic anemia not only
by eating a dishful of broad beans, but even by walking through
a field when the plants are in flower. Thus, the responsible agent
for Favism is present in the pollen as well as in the bean itself
as Rosen and Scanlan (1948) have observed.

The disease itself has been recognized for centuries. Its known
distribution is limited to peoples of Mediterranean origin, to
Spaniards, Italians, Greeks, Armenians and Jews. But the inci-
dence of Favism is unknown because of varying susceptibility.
An individual, not previously affected, may develop symptoms
unexpectedly after eating the broad beans. Recent studies in
Israel show that Favism is inherited as a sex-linked trait.

Similarity between Favism and the drug-induced hemolytic
anemias suggested a common biochemical mechanism. In Italy
Sansone and Segni (1957) have shown that blood glutathione
levels in subjects predisposed to Favism diminished during the
episodes characteristic of the disease. In Israel, Szeinberg *et al.*
(1958) similarly confirmed the evidence that Favism is associ-
ated with glucose-6-phosphate dehydrogenase deficiency. In the
Fava-prone individual blood enzyme levels may be materially
decreased during an attack.

Under investigation is the relationship between Favism and
primaquine-sensitivity, the question being whether the same gene
is involved or whether this represents yet another biochemical
polymorphism. Of great interest is the fact, observed by Motul-
sky (1960) that Favism also appears to be associated with malar-
ial areas. It is, interestingly, characteristic of Sephardic and not
Ashkenazic Jews.

Clearly, malaria has left its impact on many populations
through numerous adaptive polymorphisms. Favism, in particular,
provides the opportunity for further studies: one would like to
know why the incidence of a primaquine-type of sensitivity is so
high in Kurdistani Jews. Assuming that the gene is inadaptive in
a non-malarious area, one should be able to compute the rate of
genetic change for numerous Mediterranean populations (Arme-

nians, Syrians, Greeks, Italians and Jews) that have migrated to northern Europe, North America and the non-malarious regions of South America.

OTHER DISEASES AND RACE

As the reader of this book undoubtedly knows, a great many diseases have been considered to be either race-limited or population-limited at various times. For a few, the evidence stands. Tay-Sachs disease and familial dysautonomia are both more common in Jews. Leprechaunism seems to be restricted to people of Irish descent. Silferskiold's disease apparently is a Scandinavian specialty. Leukemia appears to be inherently more common in Japanese, a matter of considerable importance in ascertaining the effects of radiation in Hiroshima and Nagasaki.

For some of the data apparently linking race and disease, social and economic explanations suffice. Alcoholism and Irish ancestry are not likely to be related on a purely genetic basis. Rheumatic heart disease has been common among each immigrant group in the United States in succession. Insanity, despite possible genetic predisposition often indicates the working of learned behavior, with immigrants adopting the typical psychotic directions of their hosts within a generation.

With other disorders such as coronary artery disease, the picture becomes more complicated. We know that the Bantu and Zulu, previously coronary-free, do develop coronary artery disease as they become Europeanized. Yemenite Jews in Israel were once coronary-free: now they resemble European Jews in their growing predisposition to coronary disease.

Diabetes too, once a "European disease" and especially common among European Jews, now turns up frequently among the detribalized natives of South Africa, among Japanese professionals and among East Indian intellectuals. To list such diseases as exclusively "hereditary" therefore does violence to observed fact, and ignores the role of nutrition.

THE ADAPTIVE NATURE OF HEREDITARY DISEASES

Nevertheless, such a disease as coronary heart disease does have a demonstrable genetic component, in the simply-inherited metabolic disorder *hypercholesterolemia. Hyperurecemia,* similarly inheritable, is one factor behind the painful disease of gout. Some proportion of diabetics are genetic diabetics, in whom the disorder may *not* appear in the absence of particular nutritional or emotional stresses.

The continuance of these obviously disadvantageous disease genes, indicates either a high mutation rate, or better, some equivalent advantage associated with the genes. Since infectious diseases have long been with us, it is reasonable to look to them for the explanation.

Besides malaria, one may suggest tuberculosis, yaws and syphilis, leprosy and elephantiasis, and bihlzaria. In addition, there are the childhood diseases—measles, mumps and whooping cough, scarlet fever, diphtheria and poliomyelitis. There are the ECHO and Coxsackle viruses, debilitating in the adult but often fatal in the newborn.

Within the past decade we have seen the rise of DDT-resistant flies and penicillin-resistant staphylococcus. Such genetic immunity need not be restricted to insects and bacteria. In fact, we have evidence that genetic immunity to toxins and bacteria does arise in man, and that some of the "hereditary diseases" represent adaptations to present or past epidemics.

SUMMARY

The investigation of hereditary diseases in different race-populations increasingly supports the contention that the genetic characteristics of every race are shaped by the environment in which it lives.

While purely chance events may be responsible for differing frequencies of very rare disease genes, gene frequencies of 0.1 and higher strongly suggest the workings of natural selection, and selection coefficients far higher than those considered plausible a fews years ago.

Because of environmental differences, a particular heterozygote may be advantageous in one area and disadvantageous in another

area, thus leading to differences between local races. However, local races from different geographical races may resemble each other in respect to particular gene frequencies, if subject to the same direction of selection.

Extrapolating from such diseases as sickle cell anemia, Mediterranean anemia, Favism and primaquine-drug-sensitivity, all race-limited hereditary disorders must now be considered as possible examples of adaptive polymorphism in man.

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VIII

RACE AND GENETIC DRIFT

Fᴏʟʟᴏᴡɪɴɢ inexorable laws of genetics, gene frequencies may be expected to remain constant from generation to generation. If the gene frequency for blood group O is 0.64 in this generation, it will be 0.64 in the next. If 18% of Englishmen have light hair today, 18% had light hair during Victoria's reign. Stability of gene frequencies, once panmixia is achieved makes possible mathematical excursions into the past, and prediction of future trends.

But gene frequenices do not always remain constant, thus changing the genetic nature of a race-population. One mechanism capable of changing gene frequencies is, of course, natural selection, which alters the balance of alleles in one direction or another. Mutation is a second mechanism that affects gene frequencies—introducing new genes into the population through mutating (i.e., changing) some proportion of the old. And a third evolutionary mechanism, called *random genetic drift* or more simply "drift" is also capable of altering gene frequencies but in a random or nondirected fashion.

Drift, in fact, is largely statistical variation—purely random fluctuation of gene frequencies from generation to generation. As any student of statistics knows, frequencies or proportions will differ somewhat in successive sub-samples as a result of sampling error, that is accidents of sampling. In successive human generations chance events may slightly lower a gene frequency, or raise it unpredictably and in no regular pattern (see Figure 18).

Characteristically, genetic drift is of minimal importance in large populations. Where the number of individuals, or better the number of individuals of breeding age is large, say 1000 or more, losses of genes in some lineages are balanced by gains in

GENERATION 1

GENERATION 2

GENERATION 3

GENERATION 4

Fig. 18. The mechanism of genetic drift. In small populations or with low gene frequencies regardless of population size, the frequency of particular genes may vary considerably from generation to generation. Some of the differences between local races may be attributed to this process.

others. In such a large population, random variations in gene frequency ordinarily will not exceed $\pm$ 1%. In small populations, however, random variations in gene frequency may loom large. With a breeding population of but 100, a 5% increase or decrease in gene frequency may easily occur from generation to generation. In extreme cases, as where a particular gene is rare to begin with or where chance variations run in the same direction for several generations, a gene may be eliminated from the population on a purely chance basis.

Moreover, such small populations as have been described are by no means small as human groups go. An Australian horde may number 400, but excluding grandparents and children, the size of the *breeding population* is less than 100. For a Bushman band, and for some Eskimo isolates the breeding population may be ten or even less. One can easily imagine the fate of comparatively rare genes in such a population—either becoming eliminated entirely, or the frequency becoming "fixed" at 1.00, due entirely to chance events.

Thus it is that some students of human population genetics, among them Gabriel Lasker and Joseph Birdsell, have been interested in random drift as explaining some differences between some isolates, differences between micro-races, and even differences between geographical races. If a gene frequency became fixed at either 1.00 or 0.00 just at the time a particular isolate began explosive population growth, such a gene frequency might characterize the descendants, ultimately numbering in the thousands. And, for rare and uncommon genes barely represented in a population of any size, random genetic drift may well account for the population differences that we know.

There is much attractiveness in genetic drift as an explanation for some part of racial differences. After all, most human populations have been small, Pleistocene populations rarely exceeded 50, equivalent to a breeding population of little more than ten. Neolithic communities of a hundred or so were equivalent to breeding populations of twenty-five. Only rarely, and not much before the present era, were populations larger than 2000 practicable and these populations were still "small" in terms of the number of breeding pairs.

As migrants migrated, they broke into small groups. The boat-loads of Polynesians viewed by Captain Cook constituted small populations, ideal for the mechanism of drift. Eskimo groups of not more than 50 wandered from Russian Siberia to American Alaska, and back again. Even the recent migrations into America have involved Lebanese clans, Irish towns, and Armenian villages. Such units, detached from the larger group, have undoubtedly been subject to genetic drift, as Bentley Glass has attempted to demonstrate for the "Dunkers," a religious isolate of German origin. "Drift" may account for some of the differences between Yemenite and Alexandrian Jews, or the Jews of North Africa and those of Poland or Lithuania.

Yet there are arguments against according drift too large a role in racial differentiation. Drift, being purely chance, does not distinguish between adaptive genes and inadaptive genes. Drift alone would pile up high gene frequencies for inadaptive genes, but such high frequencies would then be whittled down by natural selection. Drift could account for some differences between adjacent populations, but not differences that are distributed in a regular way forming "clines." Only for perfectly neutral genes could drift operate alone to bring about major differences, and at the present time we are increasingly skeptical of such neutral genes, as is evident throughout this book.

Nevertheless, drift could operate to lose a gene completely, and this may be the explanation for the virtual absence of blood group B in the Americas. Alternatively, drift in conjunction with natural selection could operate to speed up the direction of evolution. Given a half dozen populations all subject to the same selective forces, the population in which drift and natural selection operated together would have the best chance of survival. In such a population the largest number of individuals would come to possess the optimum gentoype, the fastest, and thus would soon outnumber the others.

Drift can now be investigated in two ways. It is possible to set up a computer program to determine the circumstances under which random genetic drift may have the maximum race-making potentiality. Alternatively, it is possible to investigate drift in small human isolates, over time, or in relation to the populations

from which they have been drawn. While these mathematical and investigative techniques can indicate the theoretical importance of drift, the extent to which drift actually has been operative in bringing about racial differences is extremely difficult to determine. Moreover, a small degree of gene-flow from the outside could well counter the direction of drift, through its effect on the size of the total breeding population.

SUMMARY

Random genetic drift, the Sewall Wright effect, most commonly called "drift" may be considered as a third evolutionary mechanism responsible for racial differentiation. Drift operates at maximum effectiveness in small populations or at very low gene frequencies. Since most human populations in the past were small, ideal in size for drift to operate, this mechanism may explain differences between micro-races, local races and even geographical races. Nevertheless drift could not operate for long in opposition to natural selection. Most likely, drift has been effective where it has coincided with the direction of selection in particular race-populations.

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* Reprinted in *Readings on Race.*

IX

RACE MIXTURE

THROUGHOUT human history countless isolates have lost their reproductive barriers and in consequence their genetic uniqueness. Remote tribes have met and exchanged members of marriageable age. Larger groups have engaged in warfare, claiming women from the defeated as concubines and slaves. In the long course of human migration dozens of aboriginal populations have been swept up in the flood-tide of population expansion, joining the winners genetically as well as politically. Victorious soldiers have not only contributed genes to the conquered nations, but through war-brides brought home have altered the genic makeup of their own groups as well.

In the days of foot-warfare and slow ox-teams and carts, admixture was largely between adjacent isolates. When the Israelites overran the Canaanites and the Philistines, they were among peoples much like themselves. Warfare among the Greek city-states or between the Romans and their Italic neighbors similarly involved micro-races or local races at most. But with the advent of well-supplied legions, with the introduction of navies, and with all means of rapid mass transport, admixture came to involve geographical races as well.

Today, we see numerous examples of crosses between disparate geographical races, with an increasingly larger proportion of the world's population so formed. Much of Central and South America is "mixed," Amerindian times European in origin, and often partly African. The American Negro is properly European times African as are the Cape Colored of South Africa. Hawaii has long encompassed European-Asiatic and European-Asiatic-Polynesian mixtures, and European-Asiatic crosses are increasingly frequent in North America now.

Race mixture, whether between local races or geographical races, must be added to natural selection and drift as a race-making mechanism. Although race mixture does not add new genes (as mutation does) and does not of itself remove genes from the population, it increases genetic variability, it results in new genotypic combinations and thus provides new grist for the evolutionary mill.

THE GENETICS OF RACE MIXTURE

Because race mixture of any kind involves a union of separate gene pools, each differing somewhat from the other, the immediate effect of admixture is to increase genetic diversity. This is least true, of course, where isolates are very like each other in gene frequencies, and most apparent where previously-separate groups differ largely in genetic makeup. In either extreme, however, increased genetic diversity provides more material for natural selection to work upon. Race mixture increases population variability and potentially at least, speeds up natural selection.

But race mixture is far more than the arithmetic addition of alleles. While making the new gene pool more complex than was true of either parental grouping, admixture also results in new genotypic combinations. As a simple example, suppose that one group contained the genes a and b, while the second group contained the genes c and d. In the hybrid population we would then find a total of ten different genotypic combinations, aa, ab, ac, ad, bb, bc, bd, cc, cd and dd. Not only is morphological variability increased as a result of admixture, but new genotypes are thus produced (Fig. 19).

The existence of these new genotypes is particularly important, because natural selection operates upon genotypes rather than on individual genes, and the new genotypes may offer advantages not present in any of the parental genotypes. By way of example, the genotypes bc and bd may ensure greater cold resistance or superior heat resistance, or they may protect the possessor against a greater range of thermal extremes. In fact, the new genotypes bc and bd may be so superior to the original genotypes aa and bb or cc and dd as to result in a marked change in the genetic nature of the hybrids. In this event the products of admixture would
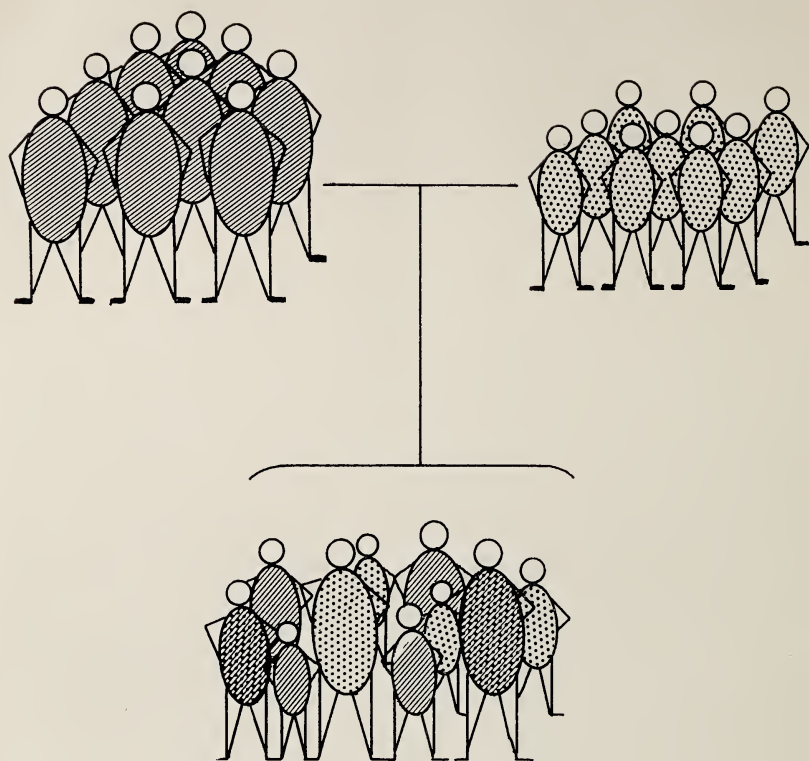
Fig. 19. Race mixture. Although race mixture does not contribute new genes it ordinarily results in new genotypic combinations not found in either parental groups as diagrammed above. See Figure 23 for an example.

eventually provide little clue as to the parental genotypes or to the proportions of each group that originally entered into admixture.

Were we to enjoy a long-term view of racial evolution in any part of the world, we would witness an almost rhythmic succession of steps. First there would be the isolates, each polished to a peak of adaptive fitness in its own ecological zone. Then there would be admixture, followed by increased genetic diversity, new genotype combinations and ultimately new peaks of fitness. Again, there would be admixture and further selection among the new genotypes, leading to new adaptive peaks again and again and

again. While we, with our birds-eye time-lapse view could see the creative power of admixture and the recurrent development of new adaptive modes, the populations involved would be conscious only of the short-term run of events. At each stage, and with a myopic view of history, they would loudly proclaim their own racial "purity."

RACE MIXTURE VIEWED AS HARMFUL

In the last century race-mixture, or more specifically mixture between geographical races was commonly viewed as harmful. Europeans, themselves the product of centuries of admixture between local races, took a dim view of the new race-hybrids their own expansion produced. Arguing on quasi-biological grounds, particularly with reference to the mule, impaired fertility was said to be one product of human racial miscegenation. Human "hybrids" of various sorts were claimed to be indolent, immoral, untrustworthy and uneducable. Progeny resulting from race mixture were alleged to fall below the intellectual level of either parent race. Somewhat inconsistent however, was the attitude toward Eurasians. As pictured in mystery novels as late as 1920, Eurasians were exceptionally sinister, sly, Machiavellian, yet capable. When a Eurasian entered an Edgar Wallace novel, shutters clanked, doors squeaked, and the heroine was tethered in a junk bound for Singapore with the pure-English hero in close naval pursuit.

Despite such novel views, human "hybrids" (that is geographical race-crosses) have evidenced no signs of impairment. Population expansion in Middle and South America belies any diminution in fertility. No evidence exists as to reduction in mental acuity. Morality among products of race mixture has been neither lower nor higher than their station in life allows. In Hawaii (as elsewhere) Eurasians have not lived up to their reputation as sinister, but have contributed lively, useful, imaginative and law-abiding citizens, many of them unusually attractive as the motion-picture audience has come to discover.

RACE MIXTURE AND HYBRID VIGOR

While exponents of racial purity were still arguing the biological inferiority of human hybrids, and were suggesting reduced fertility, impaired viability and psychological instability as the price of miscegenation, plant experimenters encountered a totally opposite trend. Intentional plant hybrids, made by crossing distinct strains or "races," proved to be superior in fertility, superior in growth and superior in disease-resistance. This phenomenon, still imperfectly understood, is the result of crossing genetically-distinct lines. Limited to hybrids, especially F_1 (first generation) hybrids, it has been termed *hybrid vigor*.

One conspicuous example of hybrid vigor is hybrid corn. In the field the stalks are taller and sturdier and the ears larger, more numerous and better filled. Hybrid tomatoes are another example: the premium "hybrid" seeds cost more, they produce bigger and more vigorous plants and finer, firmer, better-fleshed tomatoes. Intentional hybrids in other lines evidence greater disease resistance, and if well-fertilized, amazing productivity. One explanation for this phenomenon of hybrid vigor is the dispersal of deleterious genes in the F_1 generation. Notably, the advantages are less obvious in the F_2 back-crosses, so that fresh hybrid seed must be used at each sowing or planting.

Does hybrid vigor exist in man, in the first filial generation from geographical race-crossings? Here we encounter an experiental problem, that of controlled conditions. Whereas hybrid and straight-line corn can be tested in the same field, under comparable conditions of temperature, rainfall and fertilizer, such experimental controls have not been possible for man. Where can we compare, except possibly in Hawaii, Asiatic European and Asiatic-European crosses under comparable conditions of nurture?

Some few human populations have been offered as examples of hybrid vigor. The tall, vigorous, but carious Pitcairners, descendants of the Bounty mutineers and their Tahitian wives, have been offered as an example of hybrid vigor in man. But these living Pitcairners are not F_1s, but complex back-crosses, primarily of one male line. In like fashion, the Norfolk islanders are not first generation hybrids. Something other than hybrid vigor, possibly natural selection, possibly adherence to a rather English way of

life, accounts for the size and ruggedness of these descendants of the Bounty studied thirty years ago by the anthropologist H. L. Shapiro.

Still, if "hybrid vigor" exists in man, we should be able to detect it under circumstances where environmental variables are well controlled. By way of example, growth studies of English and English-Negro infants in Liverpool orphanages may well provide information on hybrid vigor. Alternatively, instead of considering first generation geographical race-crosses we may investigate highly inbred human lines, where suppressor genes may limit size and growth. Then, by measuring the progeny of out-marrying members of such isolates, size increment due to "heterosis" may well be demonstrated.

Such an investigation has been conducted by Dr. Frederick S. Hulse. For his "inbred" population, he used members of isolated Swiss cantons where cousin marriage was common. His "outbred" comparison group involved members of the same cantons who married outside of the canton. And, as shown below, the progeny of the out-marrying Swiss proved larger than did the children of the still-inbreeding Swiss. The experimental design, using intra-canton and inter-canton matings, shows how environmental circumstances can be kept relatively constant, thus avoiding the major pitfall of most attempts to uncover hybrid vigor in man.

BODY SIZE IN PROGENY OF ENDOGAMOUS AND
EXOGAMOUS SWISS MATINGS*

Measurement	Exogamous (Inter-canton)	Endogamous (Intra-canton)
Stature (cm)	168.5	166.2
Weight (kg))	73.4	72.0
Shoulder breadth (cm)	38.8	38.7
Head length (mm)	189.0	187.5

* From Hulse, F. S.: Exogamie et heterosis. *Arch. Swisses d'Anthropologie generale,* 22: 103-125, 1957.

STUDIES ON RACE MIXTURE

Though there are obvious difficulties in the study of race mixture, chiefly arising from the lack of controlled conditions, there is much to be gained from such investigations, particularly in the area of human genetics. The mode of inheritance of many human traits can best be investigated in hybrid progeny. Studies of F_1 and F_2 hybrids can help to indicate the degree of complexity of polygenic traits such as skin color, hair form, body proportions and the like. Such a characteristic as the tight spiral-tufts of Bushmen and Hottentots can not be analyzed genetically in Bushmen or Hottentot, all of whom have it, or in Europeans who lack it. But in various Hottentot-Boer "crosses," the genetics of spiral-tuft hair form can best be tackled.

Thirty years ago Caroline Bond Day collected photographs, hair samples and pedigrees of Negro-white families. Her work helped to indicate the genetic complexity of hair form, and to show that skin pigmentation was controlled by multiple genes, involving at least three loci. Studies on (aboriginal) Australian-European crosses and Asiatic-Australian crosses have further illuminated the phenomenon of graded dominance, with a particular gene acting as a dominant in one type of crossing, and as a recessive in another type of crossing. Human hybrids serve a useful purpose in the analysis of many genetic traits, and make up in part for our inability to institute experimental matings of the kind useful in plant and animal genetics.

Hybrid populations also afford the possibility of searching for linkage, something far less practical in old-established groups where linkage has been disrupted by crossing-over of chromosomal parts. The geneticist David Rife has conducted studies of this kind, in East-African populations of known and recent hybrid origin.

Finally, hybrid populations afford the possibility of calculating the degree of admixture, a useful accomplishment since historical sources are rarely quantitative. Available evidence, as investigated by D. F. Roberts and others, gives approximately 20% of "European" genes to the American Colored population. However, and contrary to popular opinion, the American Colored population contains few Amerindian genes, as Bentley Glass has demon-

strated. Thus, the myth of considerable Amerindian admixture is exploded. Clearly such studies as those by Roberts or Glass prove the value of investigating geographical race hybrids (see also Fig. 20).

HYBRIDIZATION, ADVANTAGES
AND DISADVANTAGES

In the last century race-mixture was viewed as totally bad, with dire consequences visited upon the hybrid progeny. Plant geneticists, however, discovered the phenomenon of hybrid vigor, thus pointing to a possible advantage of hybridization though such has by no means been conclusively demonstrated in man. What can we say now about the advantages and disadvantages of hybridization, or race-crossing in *Homo sapiens?*
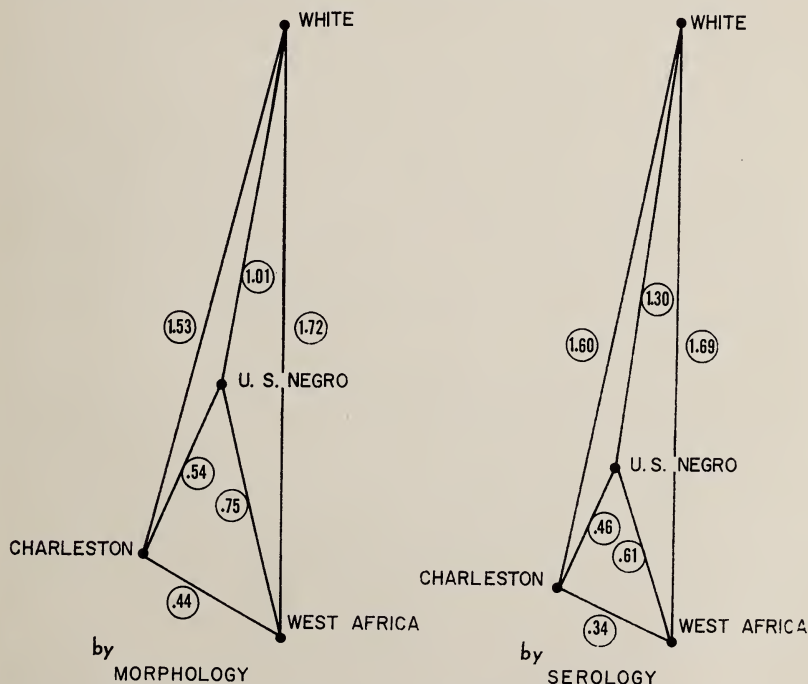
Fig. 20. Mathematical analysis of race mixture. Serological and morphological data give slightly different results when the number of variables contrasted is small. With a large number of variables the two methods would be expected to yield identical results. (From Pollitzer, 1958.)

Taking the long view and centering our attention on the population, hybridization may be considered as advantageous. By increasing genetic variability, there is a greater range of genotypes to work from, a greater likelihood of adaptive genotypes and far better prospects for long-term survival. In the long run, the more different genes the better, and race-crossing enhances genetic diversity. A hybrid race has superior long-range prospects.

The short-range view, however, centering attention upon individuals rather than the population can be somewhat different. If a population is optimally adapted to the environment, the introduction of new genes will lower average individual fitness, until the balance is restored by natural selection. But this answer itself needs qualification and emendation, as shown in the following example.

Take an East African population constantly beset by malaria and with a high incidence of the sickling gene. This population is optimally adapted to its circumstances. Admixture with non-sickling peoples would result in decreased average fitness, until such time as the balance is restored by selection. For this particular example, admixture is bad for the *individuals* concerned.

If we move this African population (or control malaria with DDT, ditch drainage and quinine) the situation may become quite different. The "hybrids" with a lower incidence of the sickling disease would exhibit increased fitness, relative to the original population.

So the "fitness" of hybrids depends very much upon circumstances. Given a population highly adapted to particular circumstances, and an intrusive population not, the hybrids would be less fit than one parental group (though more than the second). Change the circumstances and the hybrids may be superior in fitness to the first group but not the second. In a third set of circumstances hybrid fitness may be superior to both parental groups.

SUMMARY

Race mixture, the coalescence of distinct micro-races, local races or geographical races is an old human accomplishment resulting in increased genetic diversity, more rapid natural selection and (usually) new adaptive peaks.

Apart from the advantages accruing to scientific investigation, race-mixture raises the problem of population and individual fitness. No hard and fast rule can be drawn. In some cases decreased individual fitness ensues, and in other cases increased individual fitness will eventuate. From a population point of view, increased genetic diversity is of ultimate value.

Dire predictions about race mixture voiced in last-century Europe have not proved correct. Decreased fertility in particular has failed to evidence itself in hybrid human populations. Hybrid vigor, suggested by plant experiments, remains to be demonstrated in man.

That race-mixture has been an important race-making mechanism goes without doubt. Studies in populations of recent, hybrid origin hold many advantages and there are many opportunities to investigate fitness under purely local circumstances.

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*Reprinted in *Readings on Race.*

X

RACE, BEHAVIOR AND INTELLIGENCE
RACE AND TEMPERAMENT

A century ago most thoughtful and learned individuals firmly believed in racial differences in temperament. The stereotypes of the past century, sometimes still reiterated as fact, painted vastly different natures for the various geographical races. Asiatics were pictured as sly, mysterious and inscrutable and Africans were described as childlike and improvident. American Indians, with some exceptions, were portrayed as stolid, stoical and humorless. The relationship between geographical race and temperament, from a 19th century European view, may be summarized by one quotation: "Black men are ruled by passion, yellow men are bound by custom, while white men are governed by law."

Even within geographical races, inborn differences in temperament were accepted as fact. It is part of our literary heritage to speak of melancholy Danes, musical Italians and volatile Frenchmen. Various nations have been accused (by various other nations) as being humorless. Undesirable characteristics have been attributed by one group to the other. The Englishman speaks of "taking French leave," while the French equivalent is to "take English leave."

To some extent, these stereotypes were based on inadequate observations, and were due to unfamiliarity with language and gesture. It is difficult to interpret the facial expressions of nationals from another country and still more difficult to operate in a totally different culture. At the same time some stereotypes are factual. Italians do gesticulate. Germans do eat wurst. And the Sioux Indians were notably inhospitable to General George Custer. Such stereotypes, however, neglect to distinguish learned

behavior from inherent nature; they do not distinguish being Chinese from speaking Mandarin, nor a hundred generations of trading experience from inherited mercantile ability.

It is, moreover, vastly interesting to see how stereotypes change with time, The noble and restrained Romans became the voluble and musical Italians without major genetic change. The patriarchal Hebrews became the peddlers and merchants despite little accretion of outside genes. The woad-painted, skin-draped barbarians near Londunum, of Augustus' time, became the chief carriers of western culture two millennia later. The same English yeoman stock gave rise to the nasal Yankees, the elegant Virginians, the backwoods Appalachians and the Texans.

Clearly, the outstanding character-traits that do distinguish groups are culturally-determined and culturally formed. Associations between occupation and nationality melt like wax with changes in social mobility, with improved education, and with altered economic status. Predilections toward criminality, initially characteristic of each immigrant group into the United States, disappear with succeeding generations. Even the pattern of criminal proclivity, characteristically different in various national groups when they first arrive, rapidly readjusts to the general norm as is clearly evident in the table on this page. Given such data, claims for true racial differences in criminal preferences rapidly dissipate.

CONVICTION RATES FOR SPECIFIED CRIMES*

Offense	Irish Immigrants	Second Generation	Native "White"
Homicide	2.3	1.0	0.5
Rape	0.0	0.3	0.7
Gambling	1.2	2.7	3.6

* From Sutherland, E. H. and D. R. Creasey: *Principles of Criminology*. Philadelphia, J. B. Lippincott, 1955.

So far, there is no evidence for racial differences in character and temperament, other than those due to cultural conditioning. The same may be said for behavior in general. With respect to psychotic behavior, however, there may well be some differences. Some of the differences noted between Italian and Irish psy-

chiatric patients may have a genic background. Differences in adrenochrome production, in serotonin levels in the brain, even in the central nervous system may explain apparent racial differences in the predisposition to particular psychiatric disorders. Such differences, however, have not been demonstrated on a racial basis, holding the way of life constant.

Still, the most profitable area for investigation would seem to lie in the autonomic nervous system. There are clear-cut, reproducible individual differences in autonomic response specificity. Individuals exhibit marked and consistent responses to various kinds of stress. Racial differences in gene-determined response patterns are very likely, especially when one considers the adaptive nature some of these response patterns must hold in nature. Racial differences in behavior therefore, may fall to the psychophysiologist to discover, and will not encompass most areas of social behavior.

RACE AND INTELLIGENCE

Much as racial differences in temperament were firmly credited in the century now past, marked hereditary and racial differences in intellectual capacity were also accepted as self-evident truths. Long before Binet's measurements of intellectual accomplishment were published in 1905, literate and technologically-advanced Europeans held low opinions of the intelligence of illiterate and technologically-simple "natives." What the natives thought of the Europeans, unfortunately is not on record.

Intelligence tests, originally developed and used as guides for the grade-placement of school children at first provided powerful support for the assumption of racial differences in intellectual capacity. Native-born Americans ranked highest. Immigrant children from northwestern Europe came next. At the bottom of the list were American Negro children. Since Binet's test demanded reading skills and many of the Negroes were largely illiterate, a substantial proportion were rated in the dull-normal, dull, borderline and subnormal categories.

With provincial opinions as to the inferior mental capacities of "Injuns," "Chinks" and "Niggers" thus apparently confirmed by the new science of psychometries, the famous Army Alpha tests of 1917-1918 came as quite a shock. In this battery of tests, Ne-

groes (as a group) fared worse than whites (as a group) as had been expected. But Negro recruits from some northern states averaged higher on the Alphas than did white recruits from some southern states. Unless the brighter Negroes had streamed north, leaving the dullards behind, and unless there had been a comparable southward migration of the least-capable whites, differences in intelligence could not be completely racial. While a greater degree of admixture might, in theory, explain the superior showing of northern Negroes, what genetic mechanism could explain the lower average scores for southern rural white recruits?

Today, it is quite clear that intelligence tests, such as the Stanford revision of Binet's, or the Wechsler-Bellevue, or the Otis, do not primarily measure inherent gene-determined potentialities. Intelligence tests are still validated against school performance; they measure a "book" kind of proficiency. Such tests do not purport to measure many of the skills and abilities that the term intelligence commonly suggests. Intelligence tests, moreover, are never culture-free. They measure in relation to a particular cultural background, placing premiums on language skills, urban living and acquired knowledge, thus down-rating rural, non-verbal or immigrant children or adults. I.Q. test scores neatly reflect the level of motivation and (except at the extremes) not inherent ability.

Quite obviously it is impractical to get meaningful comparative intelligence scores for many preliterate peoples. Even the culture-free test is a misnomer, being a culture-constant test, or else an impossibility. Comparing or equating test scores for a Chinese peasant boy from Langsai, and a merchant's son from London introduces extraordinary problems. Only where education, opportunity and the way of life are uniform across racial barriers can meaningful comparisons be made. Possibly comparative testing can be accomplished in Hawaii. Possibly multi-racial orphanages may yield the critical data, though institutional conditions rarely afford the child an adequate opportunity to develop the skills most highly rated on the tests.

Racial differences in measured intelligence thus remain neither proven nor disproved. There are differences, but like stature, they do not necessarily indicate the maximum level of capacity in the absence of standard or controlled conditions. To the confirmed

believer in racial differences in intelligence, we can simply say that the more nearly two groups are matched in educational level, family background, opportunity and security, the closer they agree on averaged I.Q. scores. To the dedicated equalitarian, the believer in no race differences, the disparate levels in the currently best-matched Negro-white comparisons stand to be refuted.

As a matter of opinion, backed by some personal experience with mental test data, one may question whether major racial differences in working intelligence could have arisen—except in remote areas—during the millennia of human evolution. For most of man's million-year existence, the way of life of one group strongly resembled the life-way of another. Life in Paleolithic England and stone-age Tanganyika were much alike. With a salubrious climate there arose population pressure and man organized to outsmart man. In less equable climes man organized to counter nature. One may question whether it took more brains to succeed in Neolithic Ireland or in the Indus Valley in the New Stone Age.

Europe, moreover, was not always the center of technological advancement, having attained that status rather late in human affairs (and even now the baton is passing). Europeans in fact, did not compete with each other on a purely intellectual basis until rather recently, but still only a fraction of a percent later than Egyptians or Mesopotamians. Besides, one may well wonder whether it takes more "intelligence" to survive in Kansas City than in the Kalahari. Only if there was long-continued differential selection for intelligence in some areas, and not in others, could we expect true racial differences in intelligence to exist.

Yet, it would be a mistake to ignore intelligence completely in considering either race or race differences. Different skills have had adaptive value in different areas, and in some cases over a respectable number of generations. Eskimo mechanical genius stands out as one example, and mechanical skill appears to be one of the "special" abilities. The exquisite form-color sense possessed by many peoples from Japan to Thailand to Burma may well represent a second example of special skills, differentially distributed with respect to race.

A very reasonable guess is that races are comparable in the sum and total of what we call "intelligence," but differ in many

interesting details. As with the autonomic response patterns that so neatly differentiate one individual from the other, race differences may exist in form-discrimination, color-sense, tonal-memory, mechanical reasoning, abstract reasoning and with other special (rather than general) aspects of intelligence. This supposition, moreover, is directly susceptible to testing.

SUMMARY

Racial differences in gesture, speech, emotionality and way of life clearly exist but these have proven to be largely differences acquired in the social matrix. With migration, acculturation or even the simple passage of time, racial "characteristics" change immensely as in the case of the rude and crude woad-painted natives of colonial Britain. While no racial differences in temperament, behavior or intellectual capacity have been firmly established as gene-determined, and while such complexes as overall behavior or overall intelligence may not differ from race to race, there is every likelihood that components of behavior may be gene-determined, with differences in frequency from population to population. Here attention may be directed to the patterns of autonomic response to stress, to differential skills and abilities and to susceptibility to emotional disorders.

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XI

A TAXONOMY FOR MAN

$\mathbf{A}$ taxonomy is a listing, an accounting and an enumeration of the larger and smaller taxonomic groupings for any life form. Ultimately, it is concerned with species within a genus, and in a specialized taxonomy, with races within the species.

For man, that is for living man, there is but one species which Linnaeus hopefully termed *sapiens*. Within this species there is a relatively small number of geographical races and these can all be listed. There is no need to set off certain geographical races as the possible result of ancient admixture. The geographical races of mankind can be described in relatively short order, there being no more than ten of them now, however many more there were three thousand years ago.

Local races, however, pose an obvious problem. There are far too many even to list in the very few pages that are available. In addition, there is the obvious fact that as many local races have yet to be completely identified as we now know. Obviously, some selection must be made, if only to acquaint the student with the kinds of populations that we define and study. But he also needs to know the basis of the selection, why particular race-populations that he may never have heard of are included, and why others (more familiar) are not even mentioned.

A LIST OF GEOGRAPHICAL RACES

As repeatedly stated in this book, geographical races are geographically delimited collections of local races. Within a geographical race overall resemblances are greater than they are between geographical races. Nevertheless, in a particular respect, resemblances may be greater across geographical barriers than

between them as, for example, in the incidence of non-tasters (cf. Fig. 10).

Before the evolutionary nature of race was clearly comprehended, non-uniformity of local races within a geographical area posed numerous problems. One obvious expedient was to select, in each geographical race, a particular local race as the "holotype" or representative type. Then, the investigators tried to explain why the remaining local races in each area failed to come up to the specifications of the holotype, an expedient that gave good exercise to the imagination but few contributory results.

However, there was never any good reason to question the ancestry of Africans who were less dark, less prognathous or less peppercorn in hair shape than the approved Forest Negro holotype, and there was no reason to set up the "extreme" Mongoloids of Northern Asia as the holotype for Asia. The important point to bear in mind, in connection with the following listing of nine geographical races, is the existence of very considerable diversity within each. The fact that local races within each geographical race differ from each other is the subject for investigation, one of the major reasons for the study of race in man.

1. The Amerindian Geographical Race consists of a large number of local populations, ranging from Alaska, Northern Canada and Labrador, to the very tip of South America (Fig. 21). Often marginal, hunting and gathering or semi-agricultural, in most areas populations remained small, and genetic isolation was frequently complete. Under these circumstances the extent of local differentiation found among the aboriginal Americans may be taken as representative of Europe and Asia at a much earlier time.

Serologically, the Americas are characterized by the low incidence (or virtual absence) of B, the generally low incidence of A (and then only A_1), by the low incidence of N, and by varying frequencies of the Diego-positive gene Di^a. Morphologically, in hair form, tooth form, eyelid form, etc., there is an obvious overlapping with Asia and to some extent Polynesia. It is not necessary to postulate neatly-spaced waves of separate peoples from different geographical races to account for American Indian polymorphism. At the same time, some part of the wave theory may

Fig. 21. Polar-projection map of the world showing the limits of the nine geographical races described in the text. In each case geographical barriers set off the race-collections.

well be correct. There were people in the Americas by the end of the ice age, and they probably contributed to the ancestry of the present Amerindians; the amount, however, is markedly open to question.

2. The Polynesian Geographical Race occupies a vast territory in the Pacific, ranging from New Zealand to Hawaii and Easter Island (Pascua). Many now-uninhabited islands still bear relics of this hardy collection of seafaring local races.

Polynesian polymorphism is marked for hair form, facial features and body build, less so for many serological factors. There

is a range of skin colors from dark to quite light, and a fair range of nose form. Individual Polynesians may be reminiscent of Malays, Australoids, even Hopi Indians—but such resemblances are no proofs of ancestral relationships.

Serologically, the Polynesians have a high N/M ratio, as is characteristic of Pacific peoples, little B, and a high frequency of Duffy. Their population movement was primarily west to east, as Captain Cook was able to confirm.

Polynesian polymorphism was explained in terms of a simple tri-hybrid origin in the days when a three-race cosmogeny was popular. However, serological data are incompatible with such a simple explanation and the genetically-naive data analysis that favored the tri-hybrid theory has since gone out of fashion.

3. The Micronesian Geographical Race occupies a series of tiny islands in the Pacific, ranging from Ulithi, Palau and Tobi (near Guam) to the Marshall and Gilbert Islands. Possessing dark skins, wavy, helical or even "frizzly" hair, the Micronesians have been alleged to be partly "Negroid" in origin.

However, the serological picture is, except for the presence of blood group B, much like the Polynesian. There is a high phenotype frequency of A, exceeding 50%, and that primarily of the A_1 subtype. N exceeds M except in the Kapinas. Largely Duffy positive (Fy^a), and entirely Diego negative, the Micronesians occupy a unique serological niche. With R_o practically absent there can be no recent contact with Africa. With a moderate frequency of B, the Micronesians are set off from Polynesia and Australia while the absence of Di^a individuals sets them off from Asia as well.

The picture, therefore, is of distinct differences from surrounding geographical races and the virtual impossibility that the morphology and serology of the short Micronesians could have come about by any simple Negro-European-Australian mixture.

4. The Melanesian-Papuan Geographical Race isolated until World War II, exemplifies the problem of using similarities to prove origins. Often owning skulls that are primitive by any name, hair that curls, twists, frizzes and occasionally noses that seem strikingly reminiscent of the Near East, they are "stone age"

peoples newly but successfully inured to jeeps and calculators
(Fig. 22).

The Papuan-Melanesian peoples are separated serologically
from the Australians to the south having much more B and
morphologically and serologicaly from the Polynesians to the
east. Isolated from the rest of the world, possibly for 20 centuries,
they exhibit a number of gene-determined diseases all of their
own (chapter VII).

5. The Australian Geographical Race constitutes a series of
local races clearly allied with the now-extinct Tasmanians. They
are big-toothed (often exceeding the classic Neanderthaloids)
and they have very long, very narrow skulls, broadest at the base.

Fig. 22. Melanesians of New Guinea showing dark skin and helical to wooly
hair. (Photograph, courtesy of Associated Press.)

Despite their generally-dark exposed-skin color, there is a moderate amount of light or red-gold hair, and male pattern-balding reminiscent of Europeans. The Australians are notable in their very high incidence of N, in the M-N series, in the very low frequency of B (which seems to be recent and adventitious). All, apparently are Duffy positive (Fy^a).

Some Australian aborigines, even when habituated to clothes, are able to withstand very low night temperatures while sleeping in the nude. This, however, is a central Australian trait, again pointing to the importance of local adaptations within each geographical area.

6. The Asiatic Geographical Race occupies continental Asia, and extends also to Japan, the Philippines, Sumatra, Borneo and Celebes. It encompasses "the little brown men" of Indonesia and southeast Asia, the ruggedly-tall (and often big-eared) Tibetans, the North Chinese, the Mongolians and many of the natives of Formosa (Taiwan).

Asia is characterized by blood group B, up to 40%, by Diego and (insofar as is now known) the excretion of BAIB. It is the continent of inner-eye-folds, broad and fat-padded malars, little body hair and sparse beards, and coarse, straight head hair and little male pattern-balding. Sexual dimorphism is often limited by European standards; and protuberant bosoms and projecting female posteriors are rare. The birth rate in Asia, however, shows how superficial these characteristics are.

Judging from local differences, Asia was once characterized by hundreds of local races. Even today, at least a hundred can easily be distinguished. Since emigration has largely come from Japan, South China and the Philippines, we are most familiar with the reduced leg/trunk ratio of people from these areas. Short legs may in fact be a characteristic of the Asiatic Geographical Race, but our samplings are weighted and not really representative.

It should be emphasized most strongly that there is considerable polymorphism in Asia, extending even to gray-green eyes in isolated groups. Moreover, so-called Asiatic or "Mongoloid" characteristics are rather common over four-fifths of the globe. For this reason, an inner eyefold, a projecting malar, 100 mu-wide hair

or a flat nasal root should not be seized upon as indication of
"Mongoloid" origins.

7. The Indian Geographical Race extends from the high Hima-
layas (from the territory of the Improbable Snowman) to the
torrid Indian Ocean. Broken into a number of local races, with
different languages and religions and including castes that are
true local races in themselves, the Indian Geographical Race
poses a major task for the excellent anthropologists of India.

To a European, many Indians look "European." Excepting skin
color, they often look like natives of the southern and eastern
Mediterranean. Hair and beard distribution, middle-phalangeal
hair patterns and parafrontal balding complete these resem-
blances.

Blood group B is often quite high in India (>35%). The Rh
negative gene (r) is low. These two facts together separate India
from Europe, and ally it more nearly with Asia. Add to this high,
often exceptionally high proportions of non-tasters, dark, some-
times very dark skin color, and such traits as the contiguous eye-
brows and the uniqueness of this group of populations stands out.

In the Indian Geographical Race (and this includes the peoples
of Pakistan, Kashmir, etc.) the system of castes both complicates
racial analysis and points to cultural influences on raciation. While
the castes in part reflect historical-political stratification, there can
be little doubt that hereditary occupations (with all of their ad-
vantages and disadvantages) afford possibilities of differential
selection within larger demographic units.

8. The European Geographical Race comprises a collection of
local races and micro-races inhabiting (in pre-Columbian times)
Europe, Western Asia, the Middle East and Africa north of the
Sahara.

Serologically, the European Geographical Race is unique in
the relatively high frequency of the Rh-negative gene. As a
geographical race it is also unique in the extent of male hirsutism,
and of male pattern-balding. In pigmentation, however, there is
considerable overlap among geographical races, though the light-
est-skinned individuals do come from Europe, primarily northern
Europe. Sexual dimorphism is often more extreme than in other

geographical races, though this dimorphism is often exaggerated by obesity.

With its wide geographical range, and considerable polymorphism with respect to skin, hair and eye pigmentation, there is undoubtedly a series of local and regional adaptations. In all probability northern Europeans are relatively cold-adapted. Several adaptations to malaria are noted in the southern and eastern ranges of this geographical race. The high proportion of nontasters in the Baltic probably represents an adaptation to available bitter-tasting antithyroid-containing foods.

Unfortunately, human taxonomy began in Europe and among some rather atypical local races. As a result, the northwest European has been used as reference standard both for modern man and for now-extinct fossils. Actually, the European Geographical

Fig. 23. Spiral-tuft form of the body hair in an American Colored individual. This is a phenotypic expression of a genotypic combination not found in either parental stock. See Chapter IX.

Race is by no means particularly "advanced," evolutionarily speaking, and undoubtedly perpetuates a fair proportion of late Paleolithic genes.

9. The African Geographical Race constitutes a collection of local races and micro-races, all indigenous to Africa north of the Sahara. At least some local races exhibit heat-adaptations. A variety of adaptations to malaria through altered hemoglobins and changed blood enzyme levels are known.

Serologically, Africa is best characterized by the Rh_o subtype which attains 70% in some areas. Diego-positive individuals on the other hand are lacking in Africa. The rare U-negative type (in the MN-S system) apparently has its homeland in Africa. Africa too, is the distributional center of the sickling trait, the Hp_2 haptoglobin type and keloid formation.

Skin pigmentation is variable throughout Africa but in some areas the epidermis and its derivatives are literally stuffed with melanin, even to the gums, whites of the eyes and enamel of the teeth. However, in East Africa and among the aboriginal inhabitants of South Africa, skin pigmentation is considerably less extreme.

Africa includes the extremes of stature, ranging from the true pygmies to the tall Nuer. Body build is also variable, and while the extreme leg/trunk ratio is common, it is by no means characteristic of all of Africa.

The pepper-corn form of the head hair, as typically seen in Bushmen, is common in much of Africa but to the north head hair is both longer and less neatly spiral or even helical. Increased numbers of sweat glands, reported in Equatorial Africa by Hiernaux may not be characteristic of the African continent as a whole.

As with the Asiatic Geographical Race there is no one population representative of the entire geographical race. Even more important, dark skin color and helical hair elsewhere in the world need not have an African origin.

A SELECTED LIST OF LOCAL RACES

In providing a listing of local races, it is obvious that a complete enumeration could hardly be given here. Even a summary tabulation of Amerindian local races would usurp the space allotted to this chapter. But ignoring local races would be most undesirable. It would leave the field to the geographical race-collections and it would give no indication of the populations that students of race actually study.

Accordingly, a brief list of 32 local races and local race-populations has been drawn up. It is obviously not a complete listing by any account. The basis of selection here is as follows:

I. Representative large local races, numbering into the tens of hundreds of millions and including numerous micro-races often distinguishable as local "types."

II. Representative small and isolated local races corresponding almost perfectly to the idealized population-isolate.

III. Representative marginal long-isolated local races that have been unaltered by admixture for millennia.

IV. Representative hybrid or mixed local races, formed by admixture among different geographical races within the last century or two.

I. Representative Large Local Races (for location, see Fig. 26).

1. NORTHWEST EUROPEAN. Comprising the peoples of Scandinavia, northern Germany, the Low Countries, to the United Kingdom and Ireland. Variable in size and pigmentation, but including a fair proportion of blondism, light eyes and skin and a high incidence of O. Including so-called "Nordics" as a chance genetic combination.

2. NORTHEAST EUROPEAN. Comprising Poland, Lithuania and Esthonia and the Great Russias. Often heavy-set with gray or gray-blue eyes. Includes the east-Baltic and other *types*.

3. ALPINE. The rounder-bodied, rounder-headed, predominantly darker peoples of the French mountains, across Switzerland, Austria and to the shore of the Black Sea.

4. MEDITERRANEAN. The peoples on both sides of the Mediterranean, from Tangier to the Dardanelles, and including the

Fig. 26. World map, showing the location of the thirty-two selected local races described in the text.

Arabian Peninsula. Multiple adaptation to malaria (in the form of Mediterranean anemia, Favism, Primaquine-drug sensitivity, etc.) are proving of considerable taxonomic value.

5. IRANIAN (or IRANO-MEDITERRANEAN). The bigger, ruggeder and beakier peoples of Asiatic Turkey, Iran and the Soviet Union, east of the Caspian.

6. EAST AFRICAN. The long-headed, usually quite linear peoples of East Africa to the Sudan, lighter, less prognathous and less broad-nosed than numbers 8 and 9.

7. SUDANESE. Darker than 6, usually not as extreme in nose form, or lip eversion as 8.

8. FOREST NEGRO. Deeply pigmented, spiral-tuft hair, considerable prognathism, lip eversion, etc. The holotype of the "Negro." The people of West Africa and much of the Congo.

9. BANTU. A recently expanding group of peoples, usually lighter in skin color than 8.

10. TURKIC. The pastoralists and oasis farmers of Central Asia, heavy-set and broad-faced.

11. TIBETAN. The taller, more linear, quite nasal peoples of Tibet, extending northward to Mongolia.

12. NORTH CHINESE. Tall, often linear, frequently with external eyefolds. North China and Manchuria.

13. EXTREME MONGOLOID. Siberia, Mongolia to the Kamchatka Peninsula. Little facial hair, heavy facial fat-padding, snub noses with depressed roots, narrow slitted eyes with marked internal eyefolds, the holotype of the "Mongoloids."

14. SOUTHEAST ASIATIC. South China to Thailand and Burma and the offshore islands. A rapidly expanding population of generally-small peoples.

15. HINDU. Light brown to dark-skinned, often resembling Mediterraneans (4), endogamous, widely spread over India.

16. DRAVIDIAN. The rugged-faced, broad-nosed, dark-skinned peoples of southern India to Ceylon, possibly related to the Australoids.

Amerindian Groups of Local Races

17. NORTH AMERICAN. Primarily taller, more rugged hunting peoples of Canada and the United States. More A_1 than in Central or South America, especially in the Hudson Bay area. Diego-positive individuals often rare as compared with 18 below.

18. CENTRAL AMERICAN. Shorter, agricultural peoples from the American Southwest to Bolivia, almost exclusively O, less than 20% Diego-positive (Di^a).

19. SOUTH AMERICAN. Primarily the agricultural peoples of Peru and Chile. Apparently a higher incidence of Diego-positive individuals and more blood type N.

20. FUEGIAN. That is the non-agricultural inhabitants of the extreme tip of South America (including the Alacaluf, Ona and Yaghan).

II. Some Isolated Small Local Races

21. LAPP. The very small-statured, small-toothed, round-headed, almost fragile-appearing fishermen and reindeer herders of the tundra and swampy areas of West Russia, Finland, Sweden and Norway, largely north of the Arctic circle.

22. PACIFIC "NEGRITO." Small-statured, dark-skinned, frizzly-haired, actually a series of local populations ranging from the Philippines to the Queensland area of Australia and, taken as a whole, a geographical race. Despite their appellation, the Pacific Negritos have no necessary connection with the African Geographical Race or even 23 below.

23. AFRICAN PYGMY. Particularly the pygmies of the Ituri Rain Forest, whose small stature merits explanation. Most likely the product of isolation and selection early in the expansion of African populations.

24. ESKIMO. Geographically restricted to the Asiatic and American far-north, and broken into extremely small populations, evidencing many physiological adaptations to extreme cold. American and Canadian Eskimos differ from both Amerindians and most Asiatics in the high incidence of non-tasters, in the near-absence of BAIB excretors, and in a low incidence of Di^a thereby suggesting long-continued isolation and natural selection.

III. Some Long-Isolated Marginal Local Races

25. AINU. The legendary "hairy Ainu" of Yezo in northern Japan, apparently the remnant of a once much larger pre-Neolithic population, and antedating the classical Japanese. While hairier, more rugged of face, and markedly different from the Japanese, North Chinese and Kamchadals, calling the Ainu "primitive whites" turns taxonomy into Procrustes bed.

26. MURRAYIAN and 27. CARPENTERIAN AUSTRALIAN. Two distinct groups of populations, one showing numerous adaptations to moderate (32° F.) night cold, and the other more nearly tropical in adaptations. Both extremely large-toothed, exceeding the Neanderthals in this respect, and with skulls resembling Neanderthals in many details.

28. BUSHMAN AND HOTTENTOT (Fig. 25). The aboriginal inhabitants of South Africa, including (1) the desert-dwelling Bushmen of the Kalahari and (2) the cattle-owning Hottentot. Less melanotic than most Africans, small-toothed and exhibiting extreme glutal fat storage (steatopygia), the extreme peppercorn, spiral-tuft hair and early loss of subcutaneous fat.

IV. Some Hybrid Populations of Known and Recent Origin

Although all living local races undoubtedly stem from hybrid origins, the extent of admixture is generally unknown and populations from the same geographical race are ordinarily involved. The following four populations, therefore, are of particular interest because (1) several different geographical races are involved and (2) because the groups entering into admixture are known. Such hybrid populations of recent and known origin are of particular utility in the investigation of genetic linkage in man.

29. NORTH AMERICAN COLORED ("AMERICAN NEGRO"). The so-called Negro population of the United States, Bermuda and Canada. Of west-African and northwest-European origin, the accretion of European genes due to continuing admixture is partially balanced by the social phenomenon of "crossing-over."

30. SOUTH AFRICAN COLORED ("CAPE COLORED"). The analogous population of South Africa including Bushman-Hottentot and Bantu genes with a variable contribution of European genes and increasingly some of East Indian origin.

31. LADINO. Southern European and southern Amerindian, but including also (in the breeding population) Amerindians who have adopted the Ladino way of life. As with the preceding two populations, there is great local diversity. Depending on local attitudes, obvious Ladino groups may acknowledge little or no Amerindian, or little or no European in their ancestry.

32. NEO-HAWAIIAN. A complex of northwest European and southern-European with Polynesian and Chinese/Japanese and Filipino. The outstanding laboratory situation for the study of human racial hybrids under optimum living conditions.

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